A CLINICAL STUDY OF PERFORATIVE PERITONITIS

A Dissertation submitted to
THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY
In partial fulfilment of the
regulations for the award of the degree of

M.S.GENERAL SURGERY (BRANCH I)

Thanjavur Medical College and Hospital
The Tamilnadu Dr. M.G.R Medical University
Chennai,India
April 2016

CERTIFICATE

This is to certify that this Dissertation entitled "**A CLINICAL STUDY OF PERFORATIVE PERITONITIS** " is a bonafide work done by Dr.S.SANTHOSH KUMAR, under my guidance and supervision in the Department of General surgery, Thanjavur Medical College, Thanjavur doing his Postgraduate course from 2013 -2016.

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DECLARATION

I solemnly declare that this Dissertation "**A CLINICAL STUDY OF PERFORATIVE PERITONITIS** " was done by me in the Department of General Surgery, Thanjavur Medical College, and Hospital , Thanjavur under the Guidance and Supervision of my Professor Department of General Surgery, Thanjavur Medical College, Thanjavur between 2013 and 2016.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University requirements for the award of M.S Degree (GENERAL SURGERY).

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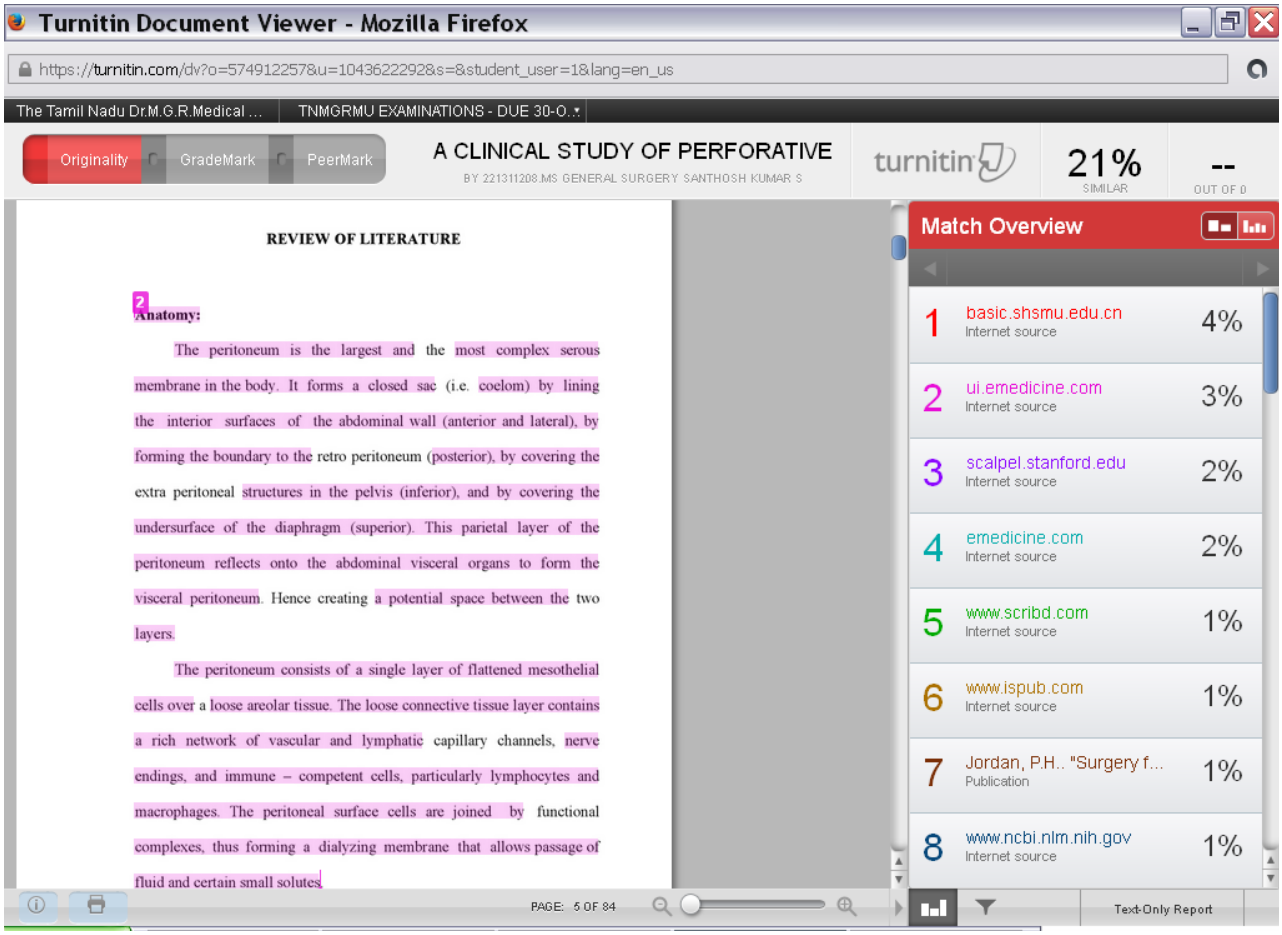
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The peritoneum is the largest and the most complex serous membrane in the body. It forms a closed sac (i.e. coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retro peritoneum (posterior), by covering the extra peritoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. Hence creating a potential space between the two layers.
The peritoneum consists of a single layer of flattened mesothelial cells over a loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillary channels, nerve endings, and immune – competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by functional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes
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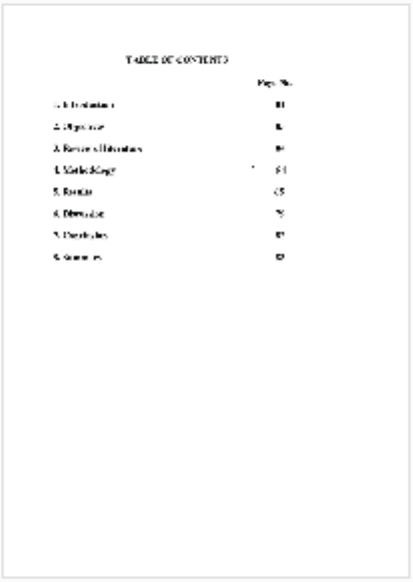

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This is to certify that The Research Proposal / Project titled

.....A CLINICAL STUDY OF PERFORATIVE PERITONITIS.....

.....

submitted by Dr. .....S. SANTHOSHKUMAR..... **of**

Dept. of .....GENERAL SURGERY..... **Thanjavur Medical College, Thanjavur**

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I thank the All Mighty God for the abundant grace & Blessings and making me successful in every field to complete the Dissertation work.

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INTRODUCTION

Peritonitis is the inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. Peritonitis is often secondary to an infection into the otherwise sterile peritoneal environment through perforation of gastrointestinal tract or a chemically irritating material, such as gastric acid from a perforated ulcer.[1] Frequent causes of secondary bacterial peritonitis include perforation due to peptic ulcer disease , acute appendicitis , ileal perforation due to typhoid & tuberculosis , jejunal perforation most often due to blunt trauma , colonic perforations secondary to closed loop obstruction or malignancy.[2]

The purpose of operative protocol is to correct the pathology while avoiding any serious accidents and to adopt a surgical procedure which is associated with minimal complications. Initial resuscitation with large volume of crystalloids , administration of broad spectrum antibiotics against gram negative bacteria and anaerobes are usually followed by laparotomy and closure of peforation.

Despite a better understanding of pathophysiology , advances in diagnosis , surgery , antimicrobial therapy and intensive care support peritonitis remains potentially fatal.

Peritonitis secondary to hollow viscus perforation is a common occurrence in this country and the spectrum of etiology in tropical countries continues to differ from western counterpart.

" In peritonitis - source control is above all

The mechanical control of the source of infection , which itself non biologic , determines the extent of the host biologic response to infection."

- *Ronald V .Maier* [3]

OBJECTIVES

1. To analyse the age / sex incidence of perforative peritonitis

2. To estimate the relative frequency of anatomical site of perforation..

3. To enlist the mode of presentation of perforation cases

4. To know the usefulness of investigative procedures in diagnosis

5. To study the outcome of surgical management for perforative peritonitis

LIMITATIONS OF STUDY

1) This study does not include the cases of traumatic perforative Peritonitis.

2) This study does not include patients with previous history of Hypertension, Diabetes, Chronic obstructive pulmonary disease.

3) Cases with previous history of abdominal surgeries were not included.

REVIEW OF LITERATURE

Anatomy:

The peritoneum is the largest and the most complex serous membrane in the body. It forms a closed sac (i.e. coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retro peritoneum (posterior), by covering the extra peritoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior).[4] This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. Hence creating a potential space between the two layers.

The peritoneum consists of a single layer of flattened mesothelial cells over a loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillary channels, nerve endings, and immune – competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by functional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes.[5]

Peritoneal Cavity:

This is the potential space between the parietal and visceral layers of peritoneum. This consists of –

- The greater sac or general peritoneal cavity.
- The lesser sac or the small omental bursa which is a diverticulum of the peritoneal cavity behind the stomach and adjoining structures.

It opens into the greater sac through a slit like aperture the epiploic foramen.

Greater Omentum:[5]

The greater omentum hangs down like a vascular apron from the greater curvature of the stomach, overlying coils of intestine. It is the most vascular part of the peritoneum, and is often called the 'policeman' of the abdomen, since it can move to a site of infection and become adherent to it, bringing protective leucocytes to the area of pathology and 'walling off' the inflammatory region.

The greater omentum consists of four closely applied layers of peritoneum enclosing blood vessels and lymphatics. The greater omentum has a continuous attachment from abdominal oesophagus to duodenum, along the greater curvature of stomach. The part of the greater omentum immediately below the stomach overlies and fuses with the transverse mesocolon .

Lesser omentum:

The two layers of peritoneum that extend from the liver onto the lesser curvature of stomach and the first inch of duodenum constitute the lesser omentum.

Peritoneal Compartments:

The peritoneum by virtue of its attachments to the posterior abdominal wall and to various viscera, divides the peritoneal cavity into compartments called

- Supracolic
- Infracolic and
- Pelvic

The Supracolic compartment is subdivided into four compartments

- Right upper or right subphrenic (sub diaphragmatic) compartment
- Right lower or hepatorenal pouch (of Morrison)
- Left upper or left Subphrenic (subdiaphargmatic) compartment
- Left lower or left subhepatic compartment.

The dividing line between the supracolic and infracolic compartments is the attachment of the transverse mesocolon to the posterior abdominal wall.

The infracolic compartment has two parts - Right and Left

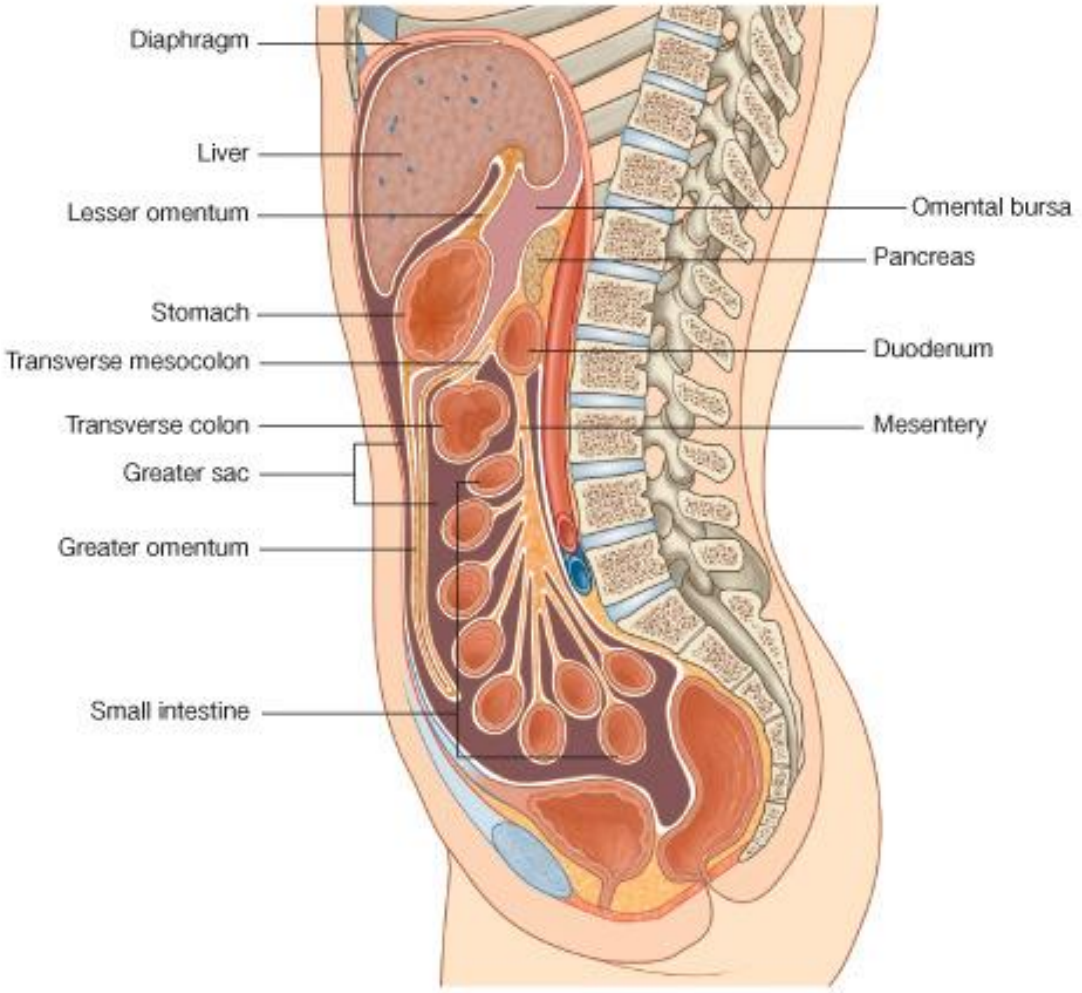


Figure 1 : Anatomy of Peritoneum

Parietal peritoneum is supplied segmentally by the spinal nerves that innervate the overlying muscles. Thus the diaphragmatic peritoneum is supplied centrally by phrenic nerve (C4) and peripherally by intercostal nerves. The remainder of the parietal peritoneum is supplied segmentally by intercostal and lumbar nerves. The visceral peritoneum has no afferent supply and pain form diseased viscera is due to muscle spasm, tension on mesenteric folds or involvement of the parietal peritoneum.

Stomach:

The stomach is the most dilated part of the alimentary tract, interposed between the oesophagus and duodenum in the upper part of abdominal cavity and lying mainly in the left hypochondriac , epigastric and umbilical region. Its mean capacity varies from 30 ml at birth, but in the adult it may accommodate upto1500 ml or more.

The junction of stomach with the oesophagus is the cardia and lies under the diaphragm, to the left of the midline at the level of T-11 vertebrae. The distal opening is the pyloric opening, at the gastroduodenal junction. It is about 1.2 cm to the right of the midline in the transpyloric plane, when the body supine and the stomach empty. The main parts of the stomach are the fundus, body and pyloric part, with the greater and lower curvatures forming the upper and lower borders and joining the anterior and posterior surfaces. Fundus is the part which projects upwards above the level of the cardia. The body extends from the fundus to the angular notch (incisura angularis) of the lower part of the lesser curvature. The pyloric part extends from the angular notch to the gastroduodenal junction, and consists of the proximal pyloric antrum which narrows distally as the pyloric canal. The circular muscle of the distal end of the canal is thickened to form the pyloric sphincter, whose position is indicated on the anterior surface by the prepyloric vein.

There are four main arteries supplying to the stomach –

- The left gastric artery arises from the coeliac axis
- The right gastric artery arises from the common hepatic artery.
- The left gastro-epipolic artery arises from the splenic artery.
- The right gastro epiploic artery from gastroduodenal artery

Veins of the same name accompany the arteries (except that there is no gastroduodenal vein) and drain into the portal vein itself or its splenic and superior mesenteric tributaries. Prepyloric vein (without an accompanying artery) drains into the portal or right gastric veins.

All lymph eventually reaches coeliac nodes after passing through various outlying groups.

- Cardiac and most of lesser curvature: left gastric nodes.
- Pylorus and distal lesser curvature: right gastric and hepatic
- Proximal portion of the greater curvature:pancreaticosplenic and nodes in splenic hilum.
- Distal portion of the Greater curvature: right gastroepipolic nodes in greater omentum and pyloric nodes .

Sympathetic fibres (vasomotor) accompanied by afferent (pain) fibers run with the various arterial branches to the stomach. The parasympathetic supply is from the vagi which control motility and secretion. The anterior vagal trunk (from the oesophageal plexuses lies in contact with the anterior oesophageal wall, usually nearer its right margin.

The anterior nerve of Latarjet, which is the termination of the left vagal trunk divides into 4 or 5 branches in a configuration that resembles a crow's foot. These terminal branches innervate the distal 6-7 cm of the antrum and pylorus. The posterior vagal trunk lies in a loose tissue a little behind and to the right of the right oesophageal margin.

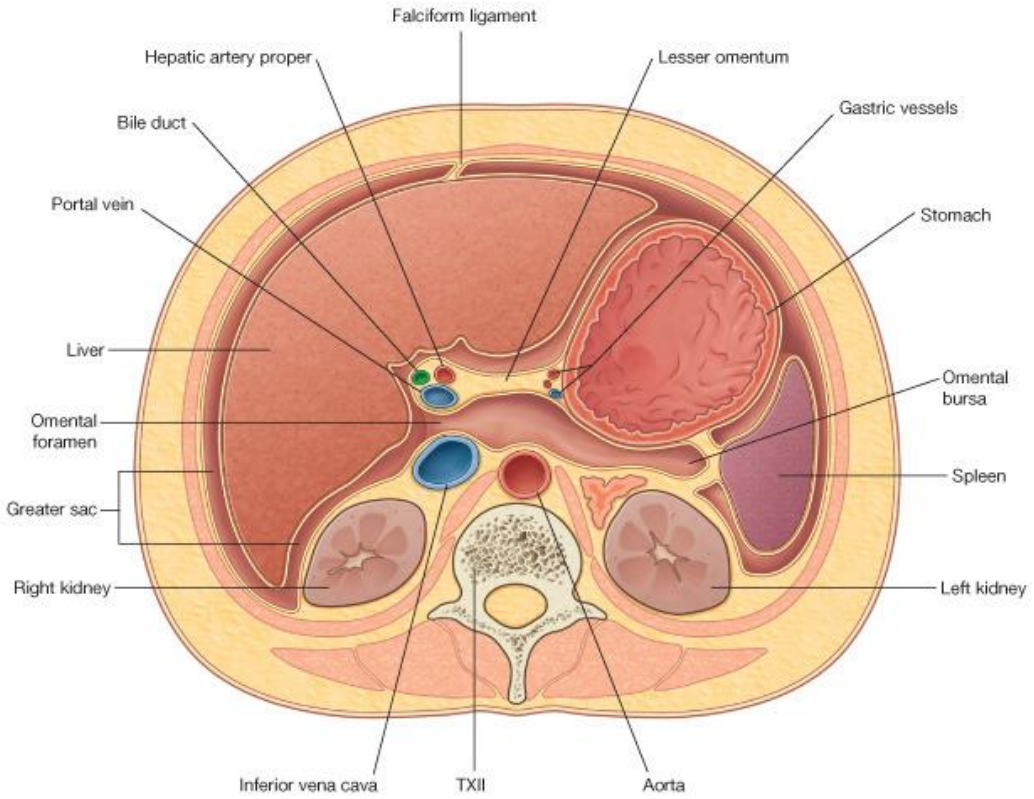


Fig 2 : Transverse section of abdomen showing the peritoneal reflection

Duodenum:

The duodenum is a C-shaped tube curved over the convexity of the forwardly projecting aorta and inferior vena cava. The first 2 cm are contained between the peritoneum, but the remainder is retroperitoneal.

It is divided into 4 parts:

- First (Superior): about 5 cm long
- Second (Descending): about 7.5 cm long
- Third (Horizontal): about 10 cm long
- Fourth (Ascending): about 2.5 cm long.

It is the peritoneal attachment which distinguishes duodenum and jejunum. The duodenum is retroperitoneal, the jejunum has a mesentry. The duodenojejunal flexure is fixed to the left psoas fascia by fibrous tissue. It is further supported by the suspensory muscle of the duodenum (ligament of Treitz).

The duodenum is supplied by the superior and inferior pancreaticoduodenal arteries, but the first 2 cm, receive small branches from a variety of sources; hepatic, common hepatic, gastroduodenal, superior pancreaticoduodenal, right gastric and right gastroepiploic.
Venous drainage drain into splenic, superior mesenteric and portal veins.
Duodenal lymph drains to coeliac and superior mesenteric nodes.

Jejunum and Ileum:

The jejunum and ileum together lie in the free margin of the mesentry. Total length varies from about 4 to 6 meters. The jejunum constitutes rather less than half the total length. The jejunum lies coiled in the upper part of the infracolic compartment, the ileum in

the lower part thereof and1 in the pelvis.

The jejunum is wider broader and thicker walled than the ileum. An ileal (Meckel's) diverticulum is present is 2% individuals, 60cm(2 ft) from the caecum and is 5 cm (2 inch) long. It represents the persistent intestinal end of the vitellointestinal duct.

Jejunal and ileal branches arise from the left side of the superior mesenteric artery and enter the mesentry. The jejunal branches join each other in a series of anastomosing loops to form arterial arcades. From the arcades straight arteries pass to the mesenteric border of the gut.

The ileal arteries form a larger series of arcades three to five in number and there is more fat in their part of the mesentry. The veins all correspond to the arteries and they drain to the superior mesenteric vein. Jejunal and ileal lymph drains to superior mesenteric nodes.

Caecum and Appendix:

Caecum is a blind pouch of the large intestine and projects downwards from the commencement of the ascending colon, below the ileocaecal junction. It's average length is about 6 cm and breadth about 7.5cm. The longitudinal muscle of the caecum is concentrated into three flat bands, the taeniae coli, between which the circular muscle layer constitutes the sacculated wall of the gut. Internally the ileocaecal junction is guarded by the ileocaecal valve.

The appendix is a worm shaped, blind ending tube varying in length 2 cm to 15 cm , which opens into the posteromedial wall of the caecum 2cm below the ileocaecal valve . The base of appendix is at the point of Convergence of three taenia coli. It may occupy one of the several Positions . It is connected by a short mesoappendix to the ileal mesentry.

Colon :

Consists of four parts: ascending, transverse, descending and pelvic colon. Of this the transverse and sigmoid are suspended in mesentery but the ascending and descending colon are plastered on to the posterior abdominal wall, so that they have posterior 'bare areas' .

Ascending Colon:

This first part of the colon, about 15cm in length extends upwards from the ileocaecal junction to the right colic flexure. Bulbous pouches of peritoneum, distended with fat, the appendices epiploicae, project in places from the serous coat.

Transverse colon:

About 45 cm long, extends from hepatic to splenic flexure in a loop which hangs down to a variable degree between these two fixed points. It is completely invested in peritoneum. The appendices epiploicae are larger and more numerous than on the ascending colon.

Descending colon:

Less than 30 cm long, this extends from the splenic flexure to the pelvic brim and in the whole of its course, it is plastered to the posterior abdominal wall by peritoneum.

Sigmoid Colon:

This extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the third piece of the sacrum. It is completely covered by peritoneum and hangs free on a mesentery – sigmoid mesocolon. The sigmoid possesses well developed appendices epiploicae.

Ascending colon and proximal two thirds of the transverse colon are supplied by ileocolic, right colic and middle colic branches of the superior mesenteric artery. Rest of the colon is supplied by left colic and sigmoid branches of the inferior mesenteric artery. The marginal artery of Drummond is the paracolic vessel of anastomosis between colic arteries from which arise the terminal arteries to colon (Vasa Recta). It lies 2.5-3.8cm from the bowel wall.The veins correspond to the arteries and reach the portal vein via the superior or inferior mesenteric tributaries.The lymph channel follows the blood vessels so that drainage is to superior or inferior mesenteric nodes. The parasympathetic supply is partly from the vagi and partly pelvic splanchnic nerves. Sympathetic supply is derived from spinal cord segments T10-L2. The pain fibres that accompany these

vasoconstrictor nerves give rise to periumbilical pain if from midgut derivatives (e.g. appendix) but to hypogastric region if from hind gut.

Physiology of the peritoneal cavity:

The peritoneum is formed by mesothelial cells, with a basement membrane supported by an underlying layer of highly vascularised connective tissue. The surface area of the peritoneum is extensive, averaging 1.8m^2 (adult male). It has been estimated that a 1mm increase in the thickness of the peritoneum can result in the sequestration of 18 litres of fluid, a fact relevant to the massive fluid shifts associated with diffuse peritonitis.[6]

Under normal condition, < 50 ml of sterile fluid is present within the peritoneal cavity – secreted from the visceral peritoneal surfaces; the fluid is circulated through the peritoneal cavity. The cephalad movement proceeds along the paracolic gutter and subhepatic spaces – due to negative pressures in the subphrenic area by diaphragmatic motion.[7] Peritoneal fluid is mostly absorbed into the lymphatic circulation via the parietal peritoneal surfaces, with the remainder absorbed through diaphragmatic lymphatics. The clearance of particulate matter, cells and microorganisms is largely dependent upon diaphragmatic lymphatics.[8] The diameter of these lymphatic stomata can be varied by diaphragmatic

stretching and contraction, from 4 to 12 microns.In addition, in the presence of inflammation the patency of stomata may be increased to augment the clearance function of the diaphragm. At inspiration, contraction of the diaphragm empties the lacunae into efferent lymphatic channels. Negative intrathoracic pressure during inspiration facilitates fluid movement into thoracic lymphatic channels, and ultimately delivered to the central circulation via the thoracic duct.[10] Following the intraperitoneal injection of bacteria, organisms can be recovered from right thoracic duct within 6 min, and from blood within 12 minutes.

A number of factors can influence this diaphragmatic clearance mechanism or "pump" –

- Blockage of the stomata by platelets or talc.
- Head up position delays appearance of bacteria in circulation.
- Reducing spontaneous respiration using general anesthesia.
- Application of positive end expiratory pressure.

Clinical observation suggests that the mortality from peritonitis is reduced in patients placed in the semi upright position – probably decreases bacterial absorption via the diaphragm. The second clearance mechanism is by phagocytosis by resident peritoneal macrophages.[11,12,13]

Local response to peritoneal infection:

The inflammatory response that occurs within the peritoneal cavity, characterized by hyperemia, the influx of fluid, recruitment of phagocytic cells and fibrin deposition. Any noxious stimulus like endotoxin associated with gram negative bacteria, gram positive bacteria, bacteroides species, irritants such as gastric juice, bile salts and meconium probably incite the inflammatory process by inciting mesothelial cell damage or direct activation of the complement system. [14,15,16] Following activation the peritoneal inflammatory process is composed of changes in blood flow, the enhancement of bacterial phagocytosis and fibrin deposition to contain or trap bacteria.[17,18]

Systemic response to peritoneal infection:

The systemic response to peritoneal infection emulates the response of the body to other forms of injury such as trauma or surgery. The development of hypovolaemia is a phenomenon central to the systemic response and probably results from the fluid influx occurring in the peritoneal cavity. The subsequent intravascular volume change leads to a reduction in venous return and cardiac output. Systemic hypotension also may be the result of the secretion of TNF, IL-1, platelet activating factor and nitric oxide. [19,20]

Diminished urine flow develops as a result of the effects of increased aldosterone and anti diuretic hormone secretion, reduced cardiac output and renal shunting of blood. This is the setting that has been dubbed as "warm" septic shock, characterized by tachycardia, fever, oliguria, hypotension and warm extremities.

Abdominal distention secondary to accumulated fluid within the peritoneal cavity – creates restriction to diaphragmatic mobility and decreases ventilatory volume, creating eventual atelectasis. The accumulation of fluid in the pulmonary interstitium and alveoli decreases pulmonary compliance and decreased work of breathing. Early manifestation is hyperventilation and the development of respiratory alkalosis. With the worsening of the pulmonary edema and alveolar collapse; severe hypoxemia will develop, creating the adult respiratory distress syndrome (ARDS). Tissue metabolism is severely altered during the response to peritonitis. Tissue hypoxia leads to anaerobic glycolysis leading to metabolic acidosis. The severe loss in the lean body mass that can occur from protein catabolism occurs rapidly and is only partially ameliorated by the use of nutritional support.

Classification, Etiopathogenesis and Pathology: [21]

Peritonitis is organized into three divisions based upon the source and nature of microbial contamination**.**

I. Primary Peritonitis

A) Spontaneous bacterial peritonitis

develops in chronically ill patients with ascites , renal failure patients on dialysis , nephrotic syndrome.

B) Primary bacterial peritonitis

Occurs in otherwise healthy people in absence of surgery or trauma and is the result of primary infection of peritoneal lining by streptococcal organisms

II. Secondary Peritonitis

A) Hollow viscus perforation

B) Post operative peritonitis

Due to anastomotic leak , stump insufficiency.

It refers to peritoneal infection arising from an intra-abdominal source, majority of these episodes are the result of primary lesions of the stomach, duodenum, small intestine, colon and appendix. It is by far the most common form of peritonitis.

III. Tertiary peritonitis :

It develops following persistence or recurrence of intraabdominal infection following apparently adequate therapy of secondary peritonitis. These patients do not benefit from laparotomy as the infection is diffuse and poorly localised . It appears to be more a reflection than a cause of an adverse outcome.

Causes of perforative peritonitis :

Stomach

- Peptic ulcer perforation
- Malignancy (e.g. adenocarcinoma)

Duodenum

- Peptic ulcer perforation

Small bowel

- Salmonella enteritis
- Intestinal tuberculosis
- jejunal diverticulosis
- Meckel diverticulum
- Intestinal ischaemia
- Incarcerated hernia (internal and external)
- Malignancy (rare)

Large bowel & Appendix -

- Appendicits
- Closed loop obstruction
- Malignancy
- Colonic volvulus
- ischaemic bowel
- Ulcerative colitis and Crohns disease
- Amoebic colitis.

History: [22]

Until the end of 19th century, the intraabdominal infections were treated nonoperatively with a mortality of 90%. Surgical principles were enunciated during the first two decades of 20th century and have been uniformily applied in the management of peritonitis since 1930.[23]

The principles which have by remain unchanged are:

I. Elimination of the source of infection .

II. Removal of infected material from peritoneal cavity. [24]

With widespread application of these principles to the treatment of peritonitis, the mortality came down to 40-50%. The further trends of decline in the mortality became visible in 1970' s and 1980's . [25] The drop is attributed to the better understanding of bacteriology of the disease,availability of powerful antibacterial agents against both aerobes and anaerobes seen in peritonitis and better understanding of organ dysfunction in sepsis and efficient ICU care. The declining trend appears to have reach a plateau with the emergence of new problems. For example the problems of microbial resistance in compromised host resulting in peritonitis which may be resistant to number of antimicrobials. Such a scenario is attended upon by a higher mortality.[26]

Bacteriology of peritonitis

The insight gained into the bacterial etiology of disease has resulted in significant advances in the antimicrobial therapy of the disease. Although most of the bacteriologic etiology of peritonitis was identified by Freidrich and Heyde in 1920s, the important role of anaerobes remained obscure to most surgeons until 1970s.

The bacteria released into the peritoneal cavity following perforation of a hollow viscus cause secondary peritonitis. The two important facts that have paramount bearing on the treatment of peritonitis are the polymicrobial nature of the infection and mixed aerobic anaerobic pathogens occurring as the commonest offending bacteriologic combination.

Antibiotic selection [27,28]

When selecting an antibiotic for the patient of peritonitis, the following consideration should be kept in mind –

1) It should be directed against the well known typical spectrum of aerobic and anaerobic organisms
2) It should achieve effective concentration in the blood and Peritoneal cavity
3) It should be safe and devoid of serious toxicities and
4) Should be backed by the results of valid clinical trials.

For most cases of community acquired bacterial peritonitis i.e. appendicitis, diverticulitis, perforated ulcer disease, monotherapy with an agent active against both aerobes and anaerobes is the preferred choice.

Peptic Ulcer disease: [29,30]

Peptic ulcer disease remains one of the most prevalent and costly gastrointestinal diseases. Elective admission has decreased dramatically while admissions for complications related to ulcer disease have shown little change. Peptic ulcer disease has decreased in men and increased in women.Although the reason for the decrease in men is unknown, it may reflect the decrease in smoking among men. It is speculated that the increase in women with peptic ulcer disease was in past due to an increase in smoking and at present due to an increase in NSAID ingestion.[31,32]

On the other hand there has been a consistent increase in the age of the population affected by perforated peptic ulcer in virtually every study worldwide.[33,34] H.pylori infection has drastically changed the understanding of peptic ulcer disease. Moreover, there is a high recurrence rate for peptic ulceration following discontinuation of medical therapy. Thus, there is a renewed interest in operative management of patients suffering from peptic ulcer disease. Although the indications for surgery have not changed dramatically over the last

several decades i.e.perforation, bleeding, obstruction, the type of operation has changed in the H.pylori era .[35,36,37] However, recent studies indicate that vagotomy may not even be necessary in some situations such as perforation of the duodenum, provided that H.pylori is eradicated.

Location and type of ulcer:

Peptic ulcer disease can be divided into gastric and duodenal ulcers. Both types tend occur near mucosal junctions. For example, duodenal ulcers usually occur at the duodenal pyloric junction, whereas gastric ulcers tend to occur at the oxyntic – antral junction, the antral pylori junction. Duodenal ulcer disease is a disease of multiple etiologies. The only absolute requirements are secretion of acid and pepsin in conjunction with either H.Pylori infection or ingestion of NSAIDS.

In comparison gastric ulcer may present in four forms –

Classification of benign gastric ulcers

Acute superficial:

Single or multiple (erosions)

Chronic:

Type I, usually lesser curve.

Type II, combined

Type III, prepyloric

Type IV, proximal stomach < 2 cm from oesophageal junction.

Pathogenesis of peptic ulcer disease :[39,40,41]

It is now believed that 90% of duodenal ulcers and roughly 75% of gastric ulcers are associated with H.Pylori infection. Three potential mechanisms for H.pylori induced gastrointestinal injury have been proposed.

1) Production of toxic products to cause local tissue injury.

2) Induction of a local mucosal immune response.

3) Increased gastrin levels with a resultant increase in acid secretion.

The gastric mucosa barrier is disturbed by the production of an endopeptidase a powerful mucolytic and by the generation of large amounts of ammonia with an increase in the epithelial surface pH. The latter alters the mucosal charge gradient, cellular permeability and epithelial Na+ K+ - ATPase activity leading to back diffusion of H+. It also causes a local inflammatory reaction in the gastric mucosa and produce chemotactic factors that attract neutrophils and monocytes.

After H.pylori infection, ingestion of NSAIDS is the most common cause of peptic ulcer disease. The increased risk of bleeding and ulceration is proportional to the daily dosage of NSAID. Consequently, the ingestion of NSAIDS remains an important factor in ulcer pathogenesis, especially in relationship to the development of

complications and death. NSAIDS increase the risk of gastrointestinal complications approx. 2 to 10 fold. NSAID ingestion not only causes acute gastroduodenal injury but is also associated with chronic gastroduodenal injury. This risk of mucosal injury and or ulceration is roughly proportional to the anti-inflammatory effect associated with each NSAID.

The presence of chronic epigastric pain is more suggestive of ulceration. The acute gastroduodenal lesions typically appear within 1-2 weeks of ingestion, whereas chronic injury typically occurs after 1 month. Again ulcer risk is dose related. In comparison to H.pylori ulcer frequently found in the duodenum. NSAID induced ulcers are more frequently found in the stomach.

The increase in perforation in the elderly might largely be due to widespread use of NSAID in this group. Corticosteroids have similarly been implicated, and the association with perforation appears just as strong.

Pathology:

Approximately 98-99% of peptic ulcer occurs in either the duodenum or the stomach at a rate of 4:1. At least 98% of peptic ulcers are located in the first portion of the duodenum or in the stomach. Most

duodenal ulcers are generally within a few centimeters of the pyloric ring. The anterior wall of the duodenum is more often affected than the posterior wall; gastric ulcers are located along the lesser curvature. Majority of individuals have a single ulcer.

In 10-20% of patients with gastric ulceration there may be coexistent duodenal ulcer. Peptic ulcers are usually round in shape sharply punched out defects in the mucosa that penetrate at least into the sub mucosa, usually into the muscularis and some times more deeply. Most are 2-4 cm in diameter; those in the duodenum tend to be smaller. The mucosal margins of the crater are perpendicular and there is some mild edema of the adjacent mucosa. Heaping up of these margins is rare in the benign ulcer but is characteristic of malignant lesion. The base of the ulcer is smooth and clear owing to peptic digestion of any exudates. Scaring may involve entire thickness of the stomach; puckering of the surrounding mucosa creates mucosal folds, which radiate from the crater in spoke like fashion.

Small intestine perforation[42,46]

Ulceration of the small intestine is a lesion of multifactorial origin. In the tropics; typhoid fever remains the commonest cause of non traumatic ileal perforation. Other causes included tuberculosis, amoebiasis, ascariasis and non-specific illness in comparison to west

where strangulation of the bowel, diverticula, and foreign bodies, Crohn's disease and radio therapy are common.[45,46,48]

Typhoid Enteritis:[50,51]

Typhoid enteritis is an acute systemic infection of several weeks' duration caused primarily by Salmonella typhi . The pathologic events of typhoid fever are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small mucosa, making their way rapidly to the lymphatics and then systemically hyperplasia of the reticuloendothelial system, including lymph nodes, liver and spleen is characteristic of typhoid fever. Peyer's patches in the small bowel become hyperplastic and may subsequently ulcerate with complications of hemorrhage or perforation. Perforation usually takes place in the 2nd – 3rd week of illness with gradual onset in comparison to that of peptic ulcer and is seen in only 2% of cases. The terminal ileum bears the brunt of the intestinal infection. Typhoid ulcer is an oval mucosal defect with the long dimension in the axis of the bowel and usually situated in the terminal ileum. There will be hyperplasia and ulceration of the Peyer's patches of the intestine, mesenteric lymphadenopathy and splenomegaly.

Tuberculosis of GIT [51]

The structures involved in abdominal tuberculosis are-

1 Peritoneum

2. Intestines

3. Mesenteric lymph nodes

Intestinal tuberculosis

Gastrointestinal tuberculosis forms the bulk of what goes by the name of abdominal tuberculosis. It occurs in two forms-

Primary :

Infection is usually caused by bovine strain of the mycobacterium. and results from ingesting infected milk. It accounts for the 10% of the reported cases.

Secondary :

This constitutes majority of the cases and occurs due to swallowing of the sputum containing bacilli by patients with active pulmonary tuberculosis. Ileocaecal and jejunoileal areas are the commonest sites of involvement because of the apparent affinity of tubercle bacilli for lymphoid tissue and a physiological stasis[53,54]

There are several mechanisms by which tubercular enteritis may occur

1. From the swallowing of infected sputum in active pulmonary tuberculosis.
2. Ingestion of contaminated milk (bovine strain)
3. By haematogenous spread from active pulmonary tuberculosis, miliary tuberculosis or silent bacteraemia during primary phase of tuberculosis.
4. Direct extension from adjacent organs (rare).

Active inflammation takes place in submucosa and serosa resulting in thickening because of edema, cellular infiltration, lymphoid hyperplasia, tubercle formation and later on, fibrosis. Mucosal ulcers multiple and transversely placed in terminal ileum, may occur as a result of endarteritis of submucosal vessel.

Gross pathological appearance has led to its traditional categorization into four forms that include are

(i) **Ulcerative** (usually in terminal ileum)

(ii) **Hypertrophic** or **hyperplastic** (usually ileocaecal region in patients with high resistance to the organisms.)

(iii) **Ulcerohypertrophic**

(iv) **Ulceroconstrictive**.

Amoebiasis:

The ulcers are described as 'bottle necked' because of their undermined edges. The ulcers have yellow necrotic floor, from which blood and pus exude. The most common sites for perforation in amoebiasis are caecum and recto sigmoid area. Usually perforation occurs into a confined space where adhesions have previously formed and a pericolic abscess results. Sometimes a sudden faecal flooding of the general peritoneal cavity occurs. Caecum is more affected than sigmoid colon.

Trophozoite enter through the crypts of Lieberkuhn and penetrate directly through the columnar epithelium by their amoeboid activity and by dissolving intestinal epithelial cells with a proteolytic ferment they secrete. They gradually burrow into the submucous coat and form colonies there. With destruction of tissues around the colonies, ulcer develops.

Ascariasis:[55]

The usual habitat of the parasite is the jejunum through it may also be found in the distal reaches of the small intestine and colon. Ascariasis by itself may not produce ulceration or perforation of the healthy gut. It usually burrows and penetrates through a pre-existing ulcer. At times, the inflammatory edema and the softening of a segment due to long leftover worm mass may lead to weak spots through which the worms extrude.

Acute Appendicitis:[56]

The inciting event in most instances of appendicitis is obstruction of the appendicular lumen. This may be due to lymphoid hyperplasia, inspissated stool (a fecolith) or some other foreign body. Obstruction of the lumen leads to bacterial overgrowth as well as continued mucus secretion. This causes distension of the lumen, and the intraluminal pressure increases. This may lead to lymphatic and then venous obstruction. With bacterial overgrowth and edema, an acute

inflammatory response ensues. The appendix then becomes more edematous and ischemic necrosis of the appendiceal wall subsequently occurs with translocation of bacteria through the ischemic wall. This is gangrenous appendicitis. Without intervention the gangrenous appendix will perforate with spillage of the appendiceal contents into the peritoneal cavity. If this sequence of events occurs slowly, the appendix is contained by the inflammatory response and the omentum, leading to localized peritonitis and eventually an appendiceal abscess. If the body does not wall off the process, the patient may develop diffuse peritonitis.

Mesenteric Ischemia:[57]

Hemorrhagic infarction is the common pathologic pathway whether the occlusion is arterial or venous .The superior mesenteric vessels are involved more frequently than the inferior mesenteric vessels, with blockage of the latter often being silent because of better collateral circulation.Damage to the affected bowel may range from reversible ischemia to transmural infarction with necrosis and perforation. Arterial insufficiency causes tissue hypoxia, leading to initial bowelwall spasm. This leads to gut emptying by vomiting or diarrhea. Mucosal sloughing may cause bleeding into the gastrointestinal tract. At this stage, little abdominal tenderness is usually present, producing the classic intense visceral pain disproportionate to physical examination findings. The mucosal barrier becomes disrupted as the ischemia persists,

and bacteria, toxins and vasoactive substances are released into the systemic circulation. This can cause death from septic shock cardiac failure or multisystem organ failure before bowel necrosis actually occurs. Bowel necrosis can occur in 8-12 hours from the onset of symptoms. Transmural necrosis leads to peritoneal signs and heralds a much worse prognosis; men might be at higher risk for occlusive arterial disease because they have a higher incidence of atherosclerosis. AMI frequently a disease of people older than 50 yrs. Young people with atrial fibrillation or risk factors for MVT, such as oral contraceptive use or hypercoagulable status may present with AMI.

Ulcerative colitis :[58,59]

The most common disease pattern in ulcerative colitis is the continuous uninterrupted inflammation of the rectal mucosa that extends to a variable distance into the more proximal colon. Inflammation in ulcerative colitis is confined to the mucosal and sub mucosal layers of the colon. There is infiltration of polymorphonucleocytes and round cells into the crypts of lieberkuhn at the base of the mucosa with multiple crypt abscesses. Confinement of the inflammation to the inner layers of the bowel wall is an important characteristic of ulcerative colitis. However, with the extensive inflammation characteristic of toxic megacolon the full thickness of the bowel wall may be involved, and the process may progress to necrosis and perforation of the colon.

Crohn's disease

Crohn's colitis is grossly characterized by a thickened colonic wall and a mucosal appearance of deep, indolent, linear ulcers, cobble stoning, friability, structuring and apthoid ulceration. Single or multiple strictures may be present in both the colon and small bowel.

Microscopically, there is transmural inflammation, sub mucosal edema, lymphoid aggregation, granulomas and ultimately fibrosis. The pathognomic microscopic feature are the noncaseating granuloma consisting of localized, well formed aggregate of epithelioid histiocytes surrounded by lymphocytes and giant cells.

Diverticular Disease:[60]

Diverticulitis is more likely if diverticula are numerous, involve much of the colon and develop at an early age. Generally only one diverticulum is involved at a time, most commonly in the sigmoid colon. Infection extends through the wall into the peridiverticular tissue, causing peridiverticulitis. As a result of the perforation (be it micro or macro) the patient may develop a pericolic abscess or sinus tract. Abscess can have a varied course – it can drain spontaneously into the colonic lumen, or erode into an adjacent structure causing a fistula, become chronic or rupture into the free peritoneal cavity.

Clinical features of perforative peritonitis :[61]

The signs and symptoms produced by the perforation vary according to the time that has elapsed since the rupture occurred. There are three stages in the pathological process that can be recognized. The symptoms of each stage can be enumerated:

Early (within the first two hours)

- Severe and generalized abdominal pain
- Hypothermia
- Pulse low and weak .
- Shallow respiration .
- Retching or vomiting (slight).
- Pain on top of one or both shoulders.

Intermediate (two to twelwe hours)

- Cessation of Vomiting.
- Decreased abdominal pain .
- Abdominal wall very rigid, tender.
- Obliteration of liver dullness.
- Severe pain on movement of the body.

Late (after twelve hours)

- Vomiting more frequent but still not profuse.
- Hippocratic facies
- Abdomen tender and distended.

- Rapid and low pulse , hypovolaemic shock
- Temperature usually elevated.

Investigations

Laboratory Studies:

A complete blood cell (CBC) count with differential count in patients with suspected peritoneal infection. Most patients with intraabdominal infections demonstrate leukocytosis (> 11,000cells /mm3) with a shift to the immature forms on the differential cell count. But patients who are immuno compromised and patients with certain types of infection (e.g. typhoid) may demonstrate absence of leukocytosis and may even demonstrate leucopenia..

Serum amylase and lipase levels in patients with possible diagnosis of pancreatitis. Urine analysis is essential to rule out urinary tract diseases (E.g. pyelonephritis may mimic peritonitis). However patients with lower abdominal and pelvic infection often demonstrate WBC in the urine and microhematuria. The presence of frank pyuria, large number of red blood cells and bacteria in the specimen suggest a urinary source of patient's symptoms.

Peritoneal fluid:

A peritoneal fluid should be evaluated for glucose, protein, and lactate dehydrogenase, and gram stain, aerobic and anaerobic culture to rule out peritoneal infection. A peritoneal fluid amylase should be done if pancreatitis or pancreatic leak is suspected; creatinine level when a urinary leak is suspected. The peritoneal levels should be compared with serum levels. Routine intraoperative peritoneal fluid cultures in defined acute disease entities (i.e. gastro-duodenal ulcer perforation, appendicitis, and diverticulitis, perforation of the colon caused due obstruction or ischemia) are controversial. Several studies have found no significant outcome in regard to postoperative complication rates or overall outcome. The antibiotic regimen is based on operative culture data in only 8-10% of the time .

Radiographs:

The presence of free, intraabdominal gas almost always indicates perforation of a hollow viscus. The commonest cause is perforation of peptic ulcer; other much less common causes are diverticulitis and malignant tumors. About 70% of perforated ulcers will demonstrate free gas, a phenomenon that is almost never seen in cases of a perforated appendix .[63,64,65] Chest films taken with the patient in an upright position are ideal for demonstrating free air because the x-ray beam strikes the

hemi diaphragms tangentially at their highest point. A lateral decubitus or even a supine radiograph is used in patients who are too ill to be moved. Left lateral decubitus views of the abdomen are also sensitive for detecting small amount of free air interposed between the free edge of the liver and the lateral wall of the peritoneal cavity. Care should be taken to include the upper abdomen, because air rises to the highest point in the abdomen, which frequently is beneath the lower ribs. Films obtained with the patient in the right lateral decubitus position are also helpful, but gas in the stomach or colon may obscure small amounts of the free air. Pneumoperitoneum can be detected in 76% of cases using an erect film only, but when a left lateral decubitus projection is included, a pneumoperitoneum can be demonstrated in nearly 90% of cases. Reasons suggested for only 76% perforations manifesting as free gas in peritoneum are sealing of perforation, lack of gas at the site of perforation or adhesions around the site of perforation.

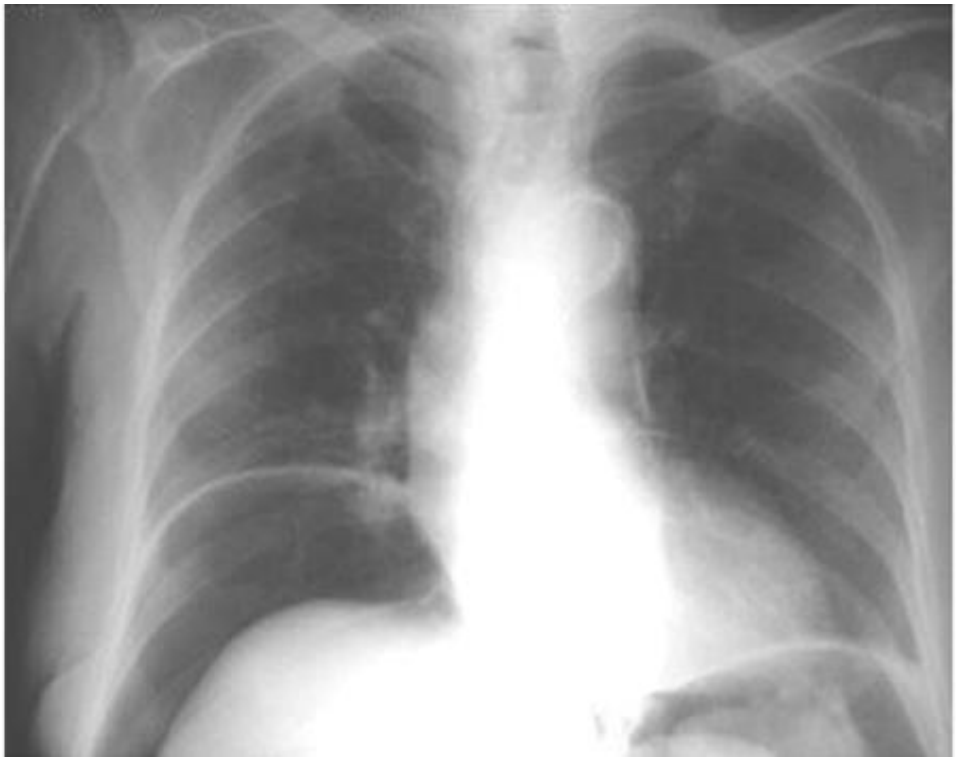


Figure 3: Radiograph showing free gas under diaphragm

Pseudopneumoperitoneum:

A number of conditions have been described which simulate free air in the peritoneal cavity i.e. pseudopneumoperitoneum. These are important because failure to recognize them may lead to an unnecessary laparotomy in search of a perforated viscus.

These are

- Chilaiditi syndrome: is distended bowel, usually hepatic flexure of the colon, interposed between the liver and the diaphragm.
- Sub diaphragmatic fat

- Distended viscus.
- Subphrenic abscess.
- Ruptured splenic abscess

Pneumoperitoneum without peritonitis:

Occasionally, asymptomatic patients or those with very minimal signs and symptoms are found to have a pneumoperitoneum.

Causes of pneumoperitoneum without peritonitis are -

i) Sealed perforation of a viscus

ii) Post operative setting

iii) Peritoneal dialysis .

iv) Post laparoscopy

Use of contrast media in suspected perforation:

Not infrequently, a patient presenting with severe upper abdominal pain has equivocal clinical signs and no free gas is demonstrable on plain radiographs. Water soluble contrast medium (about 50 ml) is given by mouth or injected through a nasogastric tube, with the patient lying on his/her right side. The patient can be examined fluoroscopically or the abdominal radiographs can be repeated after the patient has remained in this position for 5 minutes. Duodenal ulcers which have perforated but

show no free gas will normally demonstrate evidence of a leak of contrast medium. Patients with pancreatitis may have an oedematous stretched duodenal loop. Ionic water soluble contrast medium should not be given if the patient's clinical state is such that there is risk of it being inhaled and causing pulmonary oedema.

Ultrasound:[66]

Ultrasound examination allows very rapid screening of patients in suspected patients, for triage of patients who are to undergo more invasive imaging testing. Visualization of an interference echo with a shifting phenomenon is a very strong indication of the presence of free air in the abdominal cavity. This interference echo can be defined as the interruption of echo transmission due to the space between the parietal peritoneum and the surface of the liver. This free air within the peritoneal cavity can be shifted by changing the patient's position. Since the distal stomach and proximal duodenum are the most frequent sites of peptic ulcer disease, focal peritonitis due to perforation usually is located in the right upper quadrant. Unlike free peritoneal fluid, this localised exudate doesn't change shape or location when the patient's position is altered .Other findings are subphrenic or subhepatic collections. Moreover ultrasound can detect ascitic fluid as little as 10 ml. Ultrasound guided paracentesis is safe and will yield a fluid aspirate in nearly 100 % compared to clinical diagnosis with a sensitivity of 58 %.

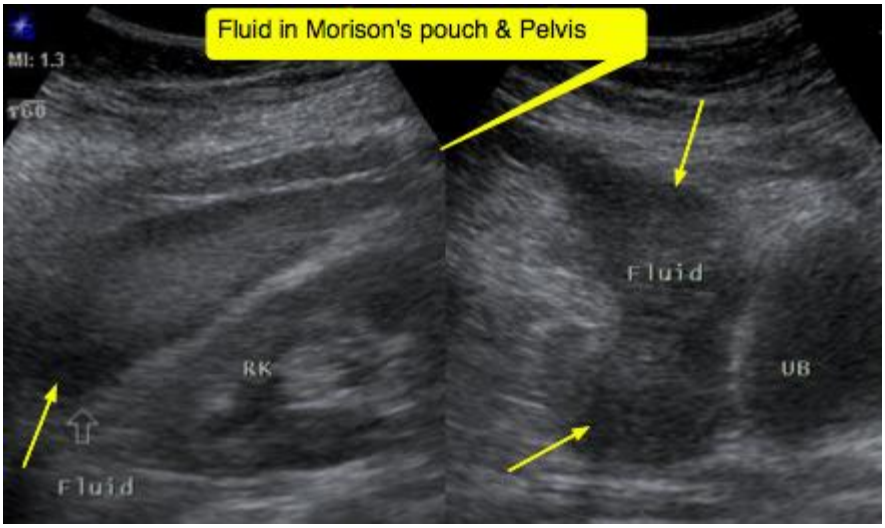


Figure 4: Ultrasound images showing free fluid in Morrison's pouch and Pelvis

Computed tomography of abdomen:[67]

The Computed tomography diagnosis of perforation was based on the direct findings of extraluminal air or gastrograffin. Indirect findings are an abscess or inflammatory mass surrounding an enterolith in the region of appendix or a bowel wall related phlegmon or abscess with fluid in the mesentery or surrounding radiopaque foreign body. Computed tomography is a valuable method in the diagnosis of alimentary tract perforation .The diagnosis can be established rapidly without patient preparation and with a high sensitivity.

Differential Diagnosis:

There are two conditions, sometimes giving rise to symptoms similar to those of perforated ulcer in which operative interference is positively contraindicated. They are

1) **Intestinal Colic:** Diagnosis usually clears on consideration of the patient's history and on careful observation of the condition of the abdominal wall, liver dullness, and the pelvic peritoneum. The radiation of the pain of biliary colic to the subscapular region and that of renal colic to the groin are sufficiently diagnostic. In ureteral stone colic, the abdominal wall is not usually rigid, and the sufferer may throw themselves in agony. After perforation of an ulcer, pain increases on movement and prevents movements. The pain of renal colic is nearly always limited to one side.

2) **Acute pancreatitis:**

Abdominal rigidity is not so generalized and constant. Cyanosis and slight jaundice are more often seen in pancreatitis, which often occur in obese patients.

Other surgical conditions that are difficult to distinguish from perforated ulcer are:

1) **Acute Appendicitis**

In the second stage perforated ulcer may be and often is

misdiagnosed as appendicitis, as the escaped contents may trickle down and cause pain in right iliac fossa. This simulates appendicitis closely, for the sequence – epigastric pain, nausea and vomiting, right iliac fossa pain, and fever – may be produced as in appendicitis, but the intensity of initial collapse and the persistence and maximal degree of tenderness over the duodenal area help to differentiate it. In appendicitis the abdominal rigidity is seldom as extensive or as marked as in perforated ulcer and the liver dullness is normal.

2) Intestinal obstruction

In their late stages, it is difficult to distinguish intestinal obstruction from perforation, for peritonitis is often a complication of late intestinal obstruction and the board like rigidity accompanying a perforated ulcer tends to diminish somewhat as the distention increases. In such cases the history and possibly the character of the vomit may serve to differentiate these conditions.

3) Ruptured ectopic gestation:

The main points in diagnosis are features of hemorrhagic shock such as the blanching of the lips, tongue, nails and the absence of true abdominal rigidity, though the abdomen is tender especially in the lower part.

4) Rupture of an ulcer with formation of localised subphrenic abscess:

Due to previous adhesions, slow leakage of the escaping gastric

contents does not flood the peritoneal cavity and the symptoms are modified. The pain may be very great but the initial collapse is not so prostrating, and the abdominal signs will soon be localised to the upper segment of the abdomen and lead to the development of a subphrenic abscess containing gas. If such an abscess develops anteriorly, the local signs of intraperitoneal suppuration are very evident, but when it is high up under the diaphragm, the signs and symptoms take longer to develop. Temperature, rigors, leukocytosis and dullness at the base of the lung consequent on pleural effusion or basal congestion will diagnose a collection of pus under the diaphragm.

Treatment:

Once the clinical diagnosis of peritonitis is made, rapid institution of both physiologic support and aggressive anti-infective therapy are imperative. *Primary objectives in the treatment of peritonitis are –*

1. Resuscitation

2. Initiation of antibiotic therapy

3. Elimination of the source of bacterial contamination

4. Reduction of the bacterial inoculum

5. Continued metabolic support.

Resuscitation:

It is an axiom that in all cases of peritonitis, some degree of hypovolaemia is present. The plasma volume must be restored and the plasma electrolyte concentration corrected. Fluid administered must contain both crystalloids and colloids. The effectiveness of fluid replacement can be judged by the normalization of pulse rate, blood pressure and mental status. Placement of a urinary drainage catheter is essential since restoration of urine output is a reliable indicator of adequate fluid resuscitation. Placement of central venous line is imperative for monitoring accurate fluid replacement.

Supplemental oxygen may be necessary and in more extreme circumstances, endotracheal intubation and mechanical ventilation may be needed to preserve oxygenation. Nasogastric decompression should be

used in the presence of an ileus to prevent pulmonary aspiration and reduce abdominal distention.

Antibiotic Therapy:

Antibiotic therapy should be initiated as soon as a clinical diagnosis of peritonitis is obtained. The initial selection of antibiotic is empiric.

The choice of antibiotics is made with the following considerations–

a) The demonstrated activity of the drug against bacteria that are presumed to be present based upon the level of gastrointestinal perforation.

b) The bactericidal activity of the antibiotic in the infected tissue.

Presumptive therapy should include coverage for both aerobic gram negative rods and anaerobic organisms. Agents that possess activity against aerobic gram negative bacilli include aminoglycoside, second and third generation cephalosporins and either ampicillin or ticarcillin combined with a beta lactam inhibitor (i.e. sulbactam or clavulanic acid).

Traditionally a 10 days therapy has been recommended, although newer studies suggest that a five day therapy may be sufficient.

Surgical Management:[69]

Surgery remains an important therapeutic modality for all cases of

peritonitis. Operative management should be directed towards the control of the source of contamination. This can be accomplished by closure of the perforation, resection of the perforated viscus, or exclusion of the affected organs from the peritoneal cavity. The secondary goal of operative management is to reduce the bacterial inoculum with the intent to prevent recurrent sepsis.

Standard intraoperative techniques to accomplish these goals include swabbing and debriding fibrin, blood and necrotic material and copious irrigation of the peritoneal cavity which are generally accepted and practiced maneuvers.[70] The use of non standard surgical techniques to prevent recurrent sepsis remains controversial. Radical debridement of fragile serosal surfaces may itself cause significant bleeding and fibrin deposition,and at present there is no perceived benefit in the use of this technique.

Peptic ulcer perforation has been classified as 'free perforation' when duodenal / gastric contents spill into the peritoneal cavity. It is called 'contained perforation' when a full thickness hole is created by an ulcer but free spillage is prevented by contiguous organs resulting in walling off.[71]

The incidence of ulcer perforation is 7-10% per 1 lakh population. Perforation is less frequent than bleeding but more common than obstruction. Pyloroduodenal perforation occurs six to eight times more

commonly than gastric perforation. Gastric perforation is more common in elderly. Prepyloric perforation and duodenal perforation occur more often in young men. 90% of perforated duodenal ulcers are seen on anterior wall. 60% of gastric perforations occur on lesser curvature and 40% are distributed all over the stomach. A recent review has shown that 52% of patients of perforation are on ulcerogenic agents.

All patients of perforation on NSAID therapy should be operated. The recurrence of ulcer perforation was reported as 7% in case of NSAID users after simple closure. The operation preferred is simple closure followed by 8 weeks of omeprazole therapy. There is no need to add definitive surgery at the time of emergency operation.

Perforated gastric ulcer tends to occur in older patients and may be associated with adenocarcinoma. This leads to higher mortality rates than the routine perforated duodenal ulcer. The operation of choice is gastrectomy as more than 10% of benign looking ulcers may be malignant.

Conservative Management:[72,73]

Most patients with peptic ulcer perforation require operative therapy on rare occasions, conservative management of perforation can be beneficial particularly in those patients who have concomitant medical illness, perforation of more than 24 hrs, systolic pressure less than 100 mm Hg at the time of admission.

These risk factors have definitive bearing on mortality rate. If one risk factor is present mortality is about 10%, if two factors are present mortality is about 40%, if three factors are present mortality is about 87%. These patients require close monitoring in intensive care unit as they may deteriorate and need operative therapy. If abdominal findings do not improve in 12 hours then operation is indicated.

Contraindications for non operative treatment

- Age > 70 years
- Steroid use
- Gastric perforation

Simple closure Vs Definitive operation:

Simple closure was first suggested for patients with gastric ulcer perforation in 1894 and later was popularized by Roscoe Graham in perforated duodenal ulcer in 1937. Longterm follow up of these patients with simple closure has significantly influenced operative management in the past 10-15 years. Simple closure will lead to satisfactory result in 1/3rd of patients. The remaining 2/3rd of patients will need acid suppression therapy or definitive operation for complications.[74] According to Boey and Wong , complications occurred in 52% of these patients (28% had bleeding, 15% had pyloric obstruction, 9% had reperforation). In this group of patients,

40% required reoperation. Ralph I George followed up 75 patients of simple closure for 5-10 yrs, 14 of these patients were on ulcerogenic drugs; 7% of them had recurrence while 6% patients who did not take ulcerogenic drugs had recurrence rate of 77%, proving that their ulcer diathesis was virulent enough to need definitive surgery. Boey and associates compared simple closure and closure with vagotomy in 78 patients with acute perforation, recurrence rate was 34% at 36 months after simple closure, reoperation was required in 43% of this group.[76] The higher reoperation rate in this group may be due to ethnic and geographic variation.

Surgical technique

The perforated duodenal ulcer closure was described by Graham.

The two principal techniques used are

a) Simple apposition of the perforation

b) Omental patch technique

Apposition should be performed using three or occasionally four sutures using suture materials such as vicryl, dexon or polydioxonone. The sutures should be through the full thickness of the duodenal wall at least 1cm from the edge of the defect. The omental patch should be used if the perforation is large or if the duodenum is so indurated that it is unlikely to hold sutures. Sutures are placed just to bring about apposition but should not be tied to approximate

the ulcer edges. Adjacent omentum should be brought up with an intact vascular pedicle. The sutures are then successively tied from the superior to the inferior side of the perforation, so as to tampon the perforation with the living omental pedicle graft.[77]

The disadvantage of sewing the ulcer shut, even if this is technically feasible, is that the omental patch placed over such a closure does not have the surface contact with the anterior duodenal serosa.

In cases of large perforation or the scarred, inflexible duodenal wall that makes simple closure difficult two options are available.

a) Conversion of the perforation into a Heineke- Mikulicz pyloroplasty.

b) Serosal patch with proximal jejunum

Laparascopic approach

Laparoscopic techniques have been applied to virtually all abdominal procedures and perforated duodenal ulcer is no exception. It was introduced by Nathanson in 1990[79]

Two approaches have been developed

a) Suturing technique

b) *Fibrin plug technique*

Pneumoperitoneum is established by either open or closed method and a 10mm trocar is inserted at the umbilicus. Exploratory laparoscopy is performed to confirm the diagnosis and to ensure that laparoscopic closure is technically feasible. Working

ports are then placed in the right hypochondrium (for grasper), left hypochondrium (for scissors, needle holder) and epigastrium (for suction irrigator). Primary closure is performed using a 5mm needle holder and a no 2-0 absorbable suture mounted on a half circle needle. The omental patch technique is performed as for open procedure.

The fibrin plug technique involves delivering solution of fibrinogen and thrombin through separate lumina of a double lumen catheter. As the two solution meet at the perforation, a fibrin plug is formed which seals the perforation.

Perfoation associated with haemorrhage:

When perforation of a duodenal ulcer in accompanied by overt gastrointestinal bleeding, a concomitant posterior ulcer should be suspected. Duodenum is opened through the anterior perforation for suture control of the posterior bleeding ulcer. An acid reductive procedure is mandatory – two alternatives being truncal vagotomy or proximal gastric vagotomy.

Definitive operations:

Truncal Vagotomy with pyloroplasty:

It has been used as definitive operation for perforated duodenal ulcer. Advantages:

i) The lesion is removed

ii) Pyloric stenosis is avoided.

iii) Length of operation is only slightly prolonged.

The transverse closure of gastroduodenostomy is performed using an interrupted one layer closure. Operative mortality of emergency truncal vagotomy with pyloroplasty for perforated ulcers varies from 0-15% in four large series since with recurrence rate of 12-15%.

Truncal Vagotomy with Hemigastrectomy:

The principal disadvantages of truncal vagotomy with hemigastrectomy are the only modest increase in operative time over truncal vagotomy with pyloroplasty, but there is an 8-10% decrease in recurrent ulceration compared with truncal vagotomy with pyloroplasty. This is preferred in cases of perforation in pre-pyloric region. The operative mortality rate for resection is extremely low in properly selected patients

Proximal gastric vagotomy : [80]

In 1973, Johnston and associates reported first clinical experience with this technique in addition to closure of perforation. Cumulative rate of recurrence was

63% after simple closure, 12% after truncal vagotomy with drainage and only 4% after proximal gastric vagotomy with simple closure in 60 patients over a period of seven years. Proximal gastric vagotomy should be avoided in patients with duodenal scarring. Jordan has suggested that all stable patients with perforated duodenal ulcer without risk factors should undergo Proximal gastric vagotomy with closure of perforation.

Post operative follow up and complications:

Perforation may be the end stage in some cases of acute ulcer perforation as in perforation caused by NSAID or ulcerogenic drugs. The patients to be put on omeprazole for eight weeks. H pylori therapy may be added to reduce the recurrence rate. In acute perforation recurrence rate was 43% and in chronic ulcer perforation was 66 to 87%. 52% may develop complications like bleeding, pyloric obstruction and reperforation. The patients with simple closure will need lifelong acid suppression agents and eradication of H pylori. NSAID, cigarette smoking and alcohol aggravate the disease.

Perforated gastric ulcer

The mortality rate of gastric ulcer perforation is higher as it occurs in older patients and is usually associated with more contamination. A biopsy should always be taken from the gastric ulcer or a partial gastrectomy performed. However, if the patient's general condition is poor, then a simple omental patch closure along with a biopsy may be adequate. Juxta pyloric ulcers behave like duodenal ulcers clinically and are treated by truncal vagotomy and pyloroplasty or by truncal vagotomy and resection.

Benign ulcers in unstable or elderly patients may be treated with excision and closure or closure with omental patch. An Ulcer high on the lesser curvature should be excised and closed. If excision is not possible,

the ulcer margin should be biopsied before closure with omental patch.

Perforated stomal ulcer : [81]

Stomal ulcers more commonly penetrate surrounding structures and occasionally perforate into the transverse colon, resulting in a gastrojejunal-colic fistula. Perforated stomal ulcers may occur many years after a simple gastroenterostomy. The most effective operation for patients with perforated marginal ulcers is to resect or reresect the stomach including the ulcer and perform a vagotomy if not done earlier. Revagotomy should be done and attention paid to find out the posterior vagus nerve, which is most likely missed. Patients with gastrojejunal-colic fistula are treated by gastric resection, vagotomy, and partial transverse colectomy.

Perforation of small intestine:

If one is dealing with perforation and associated peritonitis that precludes safe primary anastomosis, a proximal stoma and distal mucous fistula are constructed in close proximity to each other but not so close as to prevent placement of a proper fitting appliance. Once the patient is back to a normal state of health, both stoma and mucous fistula are taken down through and abdominal incision connecting both ends of the bowel. The latter are mobilized and an anastomosis is performed outside the peritoneal cavity. The bowel is then replaced in the peritoneal cavity,

the fascia closed, and the skin and subcutaneous tissue left open

Typhoid Enteritis:[82,83]

Treatment of typhoid fever and uncomplicated typhoid enteritis is accomplished by antibiotic administration. Complications requiring potential surgical intervention include hemorrhage and perforation. Intestinal perforation through an ulcerated Peyer's patch occurs in approximately 2% of cases. Typically, it is a single perforation in the terminal ileum. Simple closure of the perforation is the treatment of choice.

With multiple perforations, resection with primary anastomosis or Exteriorization of the intestinal loops may be required. According to Ameh E.A. segmental resection seems to be best treatment for typhoid perforation.

Appendicular perforation:

Generalized peritonitis following perforative appendicitis is the major cause of continuing mortality from appendicitis. This entity requires vigorous treatment. Appendicectomy must be performed in children whether the peritonitis is diffuse or not, since the other course is associated with a prohibitive mortality. But the management of this problem in adults remains acontroversy. In patients with diffuse peritonitis after perforative appendicitis appendicectomy is the treatment, as the perforation remains a continuing source of peritoneal contamination. At operation for free perforation, visualization of all peritoneal surfaces is essential. All purulent and feculent material

should be removed and dependent collection of pus should be aspirated, the peritoneal cavity should be repeatedly rinsed with warm saline solution .

Intestinal perforation in tuberculosis:

Surgery is the treatment of choice. Early surgery and anti-tubercular treatment are life saving.

(a) Simple closure of perforation: It may be done in two layers using nonabsorbable sutures. As tuberculosis strictures are short, it is a quicker treatment for those who are critically ill. Oval excision of the perforated area with transverse closure, reinforced by an omental patch may also be done. It is contraindicated when the stiches are liable to cut due to much of granulation tissue and caseation , or there is a distal stricture

b)Simple closure with bypass of strictures: Simple closure and the bypass of strictures by ileoileostomy or ileotransverse colostomy safeguard the closure against a blow out. Even when bypass is added, fistula formation frequently occurs.

c) Resection of perforated segment: If the disease is limited to a short segment and the patient is fit, the most effective treatment is the resection of the diseased segment. The segment is resected and continuity restored by end to end anastomosis.

c) Perforation at the ileocaecal region: Here closure with ileotransverse anastomosis is the preferred treatment but if the patient is fit a local ileocaecal resection can be performed

In a study by Talwar et al., the mortality rate was 29.3%. Adverse prognostic factors were operation beyond 36 hours, multiple perforations and faecal fistula formation. Mortality was least with early resection and end – end anastomosis of the perforated bowel segment.

Perforation in Diverticular disease of small bowel:

Small bowel diverticular disease is an uncommon clinical entity. Both acquired and congenital diverticula are frequently asymptomatic and become symptomatic when complicated by infection, perforation, obstruction or hemorrhage.[84]

Duodenal Diverticula:

Duodenal diverticula may be acquired or congenital. Perforation may be secondary to diverticulitis or iatrogenic following endoscopic retrograde cholangiopancreatography. It commonly occurs in the retroperitoneum over the right kidney and posterior to the head of the pancreas and duodenum. When a perforation is suspected, computed tomographic scan of the abdomen with oral and intravenous contrast is very accurate in confirming the diagnosis and in defining the extent of inflammatory reaction. Prophylactic resection of an asymptomatic diverticulum is not recommended. In the absence of significant retroperitoneal contamination, primary excision of diverticulum with two layer closure is done. In the case of large duodenal defect, serosal patch technique or a Roux-en-Y duodenojejunostomyis preferred .

In the presence of a perforation with significant edema and

contamination, a duodenal diverticulization (e.g. gastrojejunostomy, closure of the pylorus, closure of the perforation, and jejunostomy feeding tube) with drainage of the surrounding area.

Jejunal and Ileal diverticula :

In the presence of diverticulitis or perforation, resection and primary anastomosis is indicated.[85]

Diverticular perforation of colon

All patients with peritonitis must undergo emergency laparotomy. Patients are resuscitated from shock with intravenous crystalloids. Electrolyte concentration is measured and deficits especially in potassium are corrected. Intravenous antibiotics are given. A solitary perforation can be managed by resection or exteriorization. The resection need not be definitive but just enough to excise the perforated segment and leave normal colon for colostomy. A mucous fistula (or Hartmann pouch) completes the procedure. Exteriorization is acceptable provided the involved colon can be brought out tension free through a separate incision in the left lower abdominal wall. The second operation to complete definitive resection and restore colonic continuity is performed in 3 months if the patient has recovered sufficiently.

Perforation in ulcerative colitis :

Perforation of the colon occurs in about 1 to 3 percent of patients with ulcerative colitis. The likelihood of perforation is highest in the initial attack of colitis, and the incidence correlates with the severity of the initial attack and the extent of involvement of the colon. The

sigmoid colon is the most common site of perforation; the splenic flexure and transverse colon are next in order. Toxic megacolon precedes perforation in only 1/3rd to 2/3rd of cases; the remaining patients perforate in the absence of recognized colonic dilatation. Corticosteroid therapy was thought to be casual factor at one time, but now disproved, but it masks the symptoms and signs of perforation once it occurs. The diagnosis of perforation is easy to make in an untreated patient – patient has diffuse abdominal pain, tenderness, rigidity, fever and leukocytosis and free air is seen on abdominal radiographs. In hospitalized patients symptoms and signs are blunted by therapeutic agents. Immediate laparotomy is mandatory in patients with proved or strongly suspected perforation.[86]

Total abdominal colectomy with end ileostomy and exteriorization of the distal sigmoid as a mucous fistula is the procedure of choice for free perforation.

Perforation in Crohn's disease:

All patients must undergo emergency laparotomy. Patients are resuscitated from shock with intravenous fluids and antibiotics. A solitary perforation can be managed by resection or exteriorization. Simple closure with proximal colostomy leaving the perforated segment inside should be avoided. Resection need not be definitive but just enough to excise the perforated segment and leave normal colon for colostomy. A mucous fistula (or Hartmann pouch) completes the procedure.

METHODOLOGY

This study has been based on the analysis of 75 cases of hollow viscous perforation admitted to Thanjavur medical college hospital . Cases fulfilling the criteria were randomly selected for the study. Exclusion criteria were peritonitis secondary to post operative anastomotic leak, Chronic obstructive pulmonary disease patients, patient undergone any previous abdominal surgeries, trauma , any chronic illness. Out of the 75 cases of peritonitis secondary to hollow viscus perforation all underwent emergency laparotomy and the site of perforation, its pathological condition post operative complications , morbidity was seen. The procedures adopted in the management were omental patch closure, simple closure, open appendicectomy, resection and anastomosis.

Each patient was examined thoroughly, after taking a detailed history. The diagnosis and examination was made with history, clinical features and X-ray abdomen erect posture or CT abdomen to support the diagnosis. A11 cases were studied as per the proforma. And the following results were concluded.

RESULTS

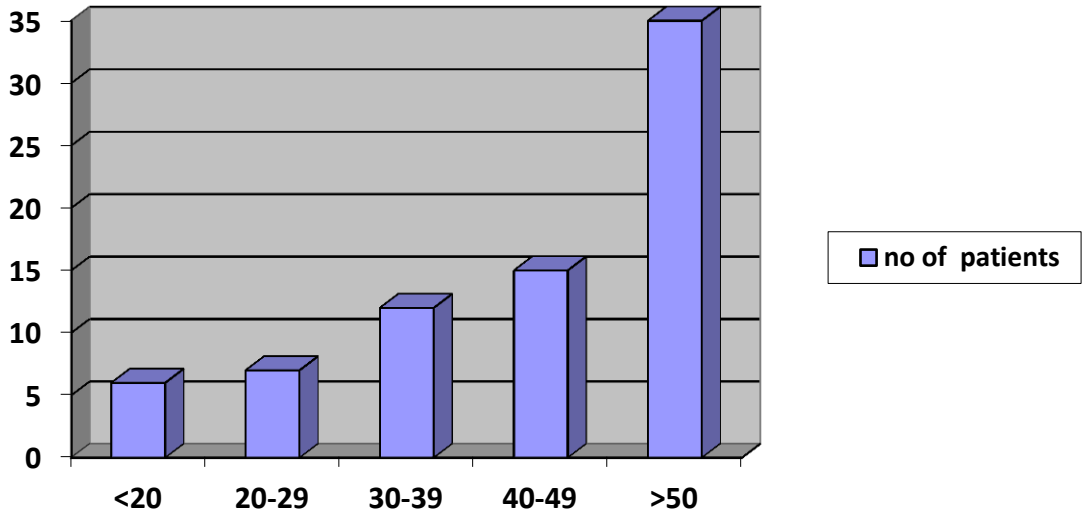
Distribution of sample by age

TABLE 1 (Distribution of sample by age)

AGE	NO. OF PATIENTS	PERCENTAGE
<20	6	8
20-29	7	9.33
30-39	12	16
40-49	15	20
>50	35	46.67

In this study most of the patients with hollow viscous perforation were above the age of 50 years. The youngest patient in this study was 14 years who was having duodenal perforation and the oldest patients was 75 years, with duodenal ulcer perforation. Perforation was found in very less frequency below 20 yrs of age.

CHART 1: Distribution By Age



Distribution by sex

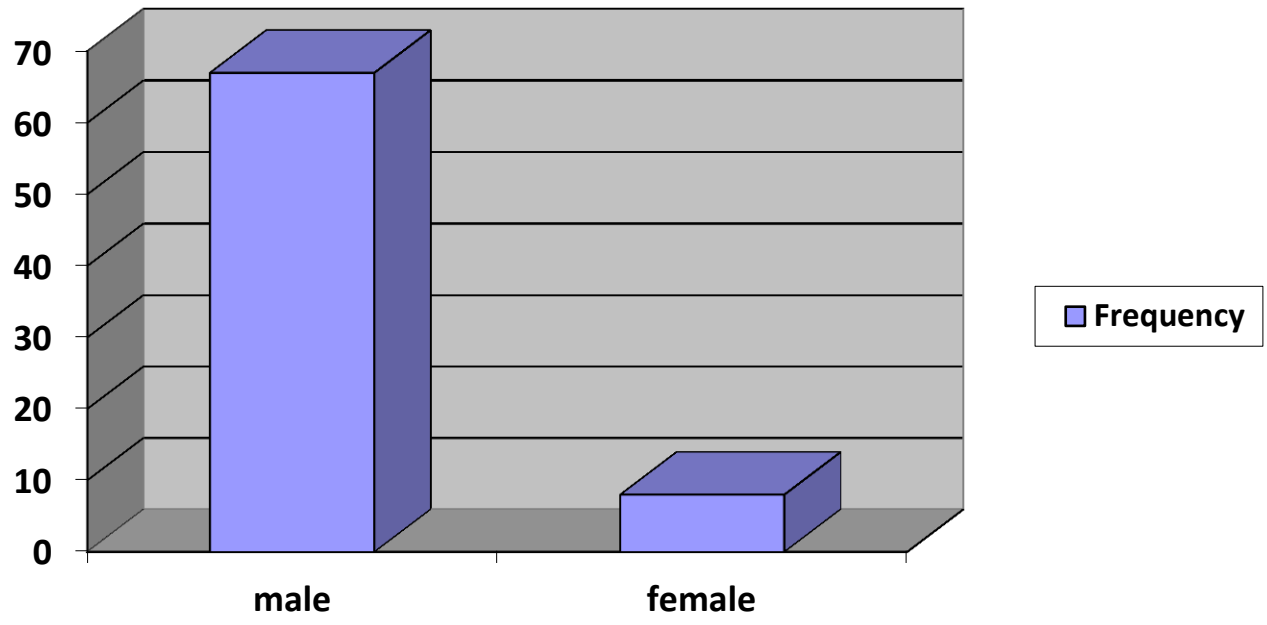
Perforation was found more commonly in males 67 cases as comparative to the females presenting in 8 patients within our sample of study of 75 patients.

TABLE 2 (DISTRIBUTION BY SEX)

GENDER	FREQUENCY	PERCENTAGE
MALE	67	89.33
FEMALE	8	10.67
TOTAL	75	100

Hence, presenting with the percentage of 89.33% as comparative to females presenting in 10.67%.

CHART 2: FREQUENCY BY SEX

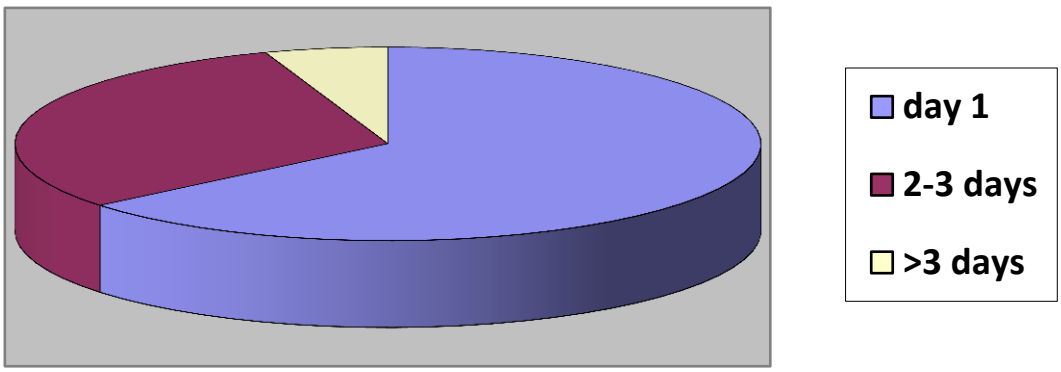


Frequency of number of days of pain in patients with perforation

Most common symptom in patients presenting with perforation was pain, present in all of the patients. The number of days with which the patients presented were quite varied depending on the time of onset of pain to the time patient came to hospital.

Most commonly the patients came to the hospital within 24hrs of onset of pain abdomen. These patients accounted for 48 patients, making it 64% of the total cases.

Chart 3 : Duration of pain



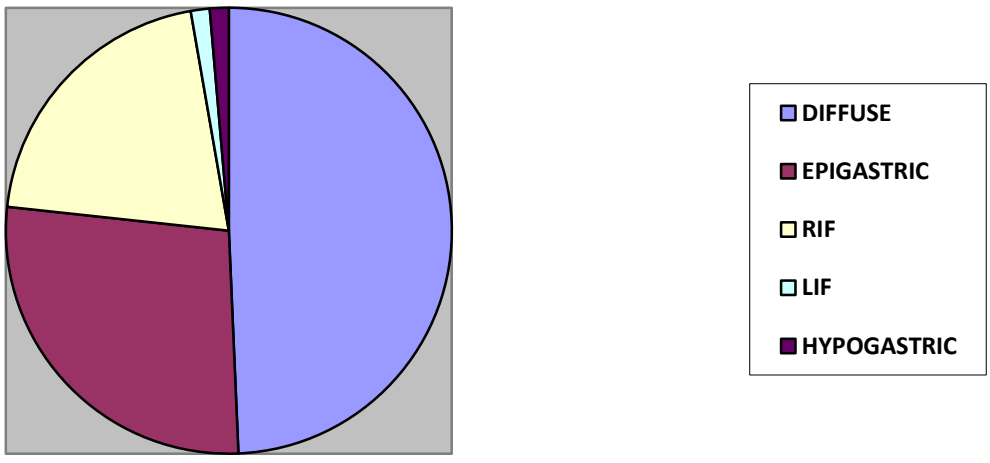
Frequency of site of pain in patients with perforation

It is as follows -Patients presenting with perforation had varied sites of pain abdomen. Most common being diffuse all over abdomen showing in 36 patients out of our sample of 75 cases, standing for 50.67% of the cases, secondly followed by pain in the epigastric region in 20 cases, standing about 26.67% of the cases.

TABLE 3 (SITE OF PAIN IN PATIENTS WITH PERFORATION)

SITE OF PERFORATION	FREQUENCY	PERCENT
DIFFUSE	36	50.67
EPIGASTRIC REGION	20	26.67
RIF	15	20
LIF	1	1.33
HYPOGASTRIC	1	1.33
TOTAL	75	100

CHART 4: SITE OF PAIN



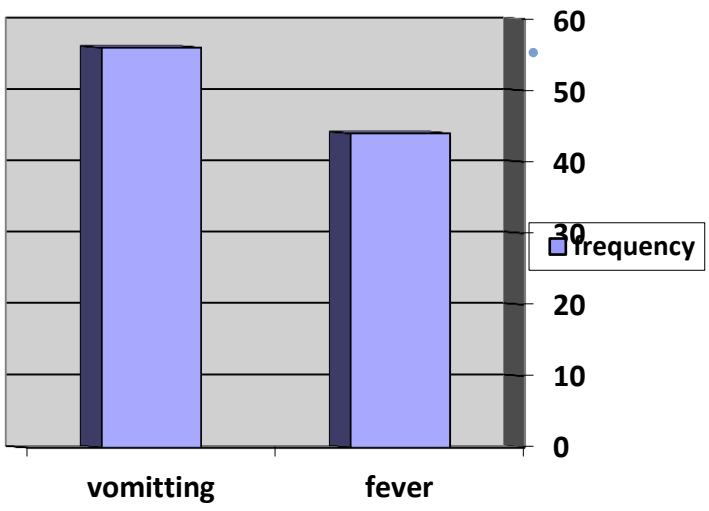
Frequency of other symptoms in patients with perforation

The other symptoms commonly present after pain abdomen were vomiting, fever and some patients also had significant earliar history which could be associated with perforation such as earliar history of acid peptic disease, fever etc. . Out of these most common after pain abdomen was vomiting, which was present in 56 patients, followed by fever which was present in 44 patients which makes 74.67% and 58.67% respectively.

TABLE 4: FREQUENCY OF SYMPTOMS IN PATIENTS WITH PERFORATION

SYMPTOMS	FREQUENCY	PERCENTAGE
Vomitting	56	74.67
Fever	44	58.67

CHART 5 : FREQUENCY OF SYMPTOMS



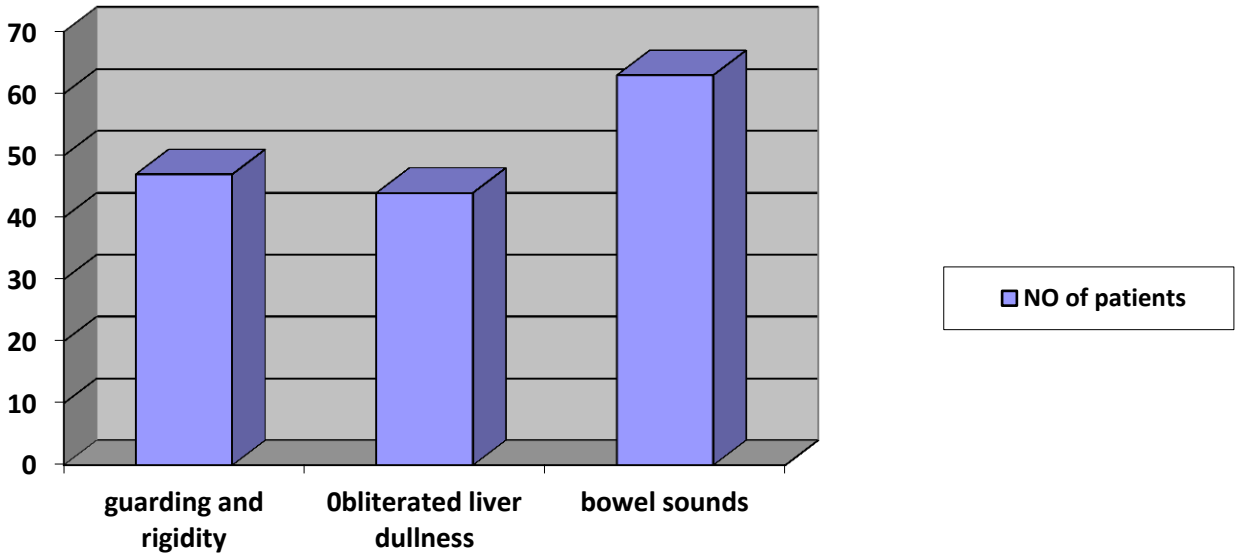
Distribution of signs in patients with perforative peritonitis

Most common sign present in almost all cases was Absence of bowel sounds which was evident in 63 cases accounting for about 84%, followed by Guarding & Rigidity which was evident in 47 cases (62.67%). This was followed by obliteration of liver dullness evident in 44 cases (58.67%)

TABLE 5 (FREQUENCY OF SIGNS IN PATIENTS WITH PERFORATION)

SIGNS	FREQUENCY	PERCENTAGE
GUARDING AND RIGIDITY	47	62.67
OBLITERATION OF LIVER DULLNESS	44	58.67
BOWEL SOUNDS ABSENT	63	84

Chart 6: Frequency Of signs



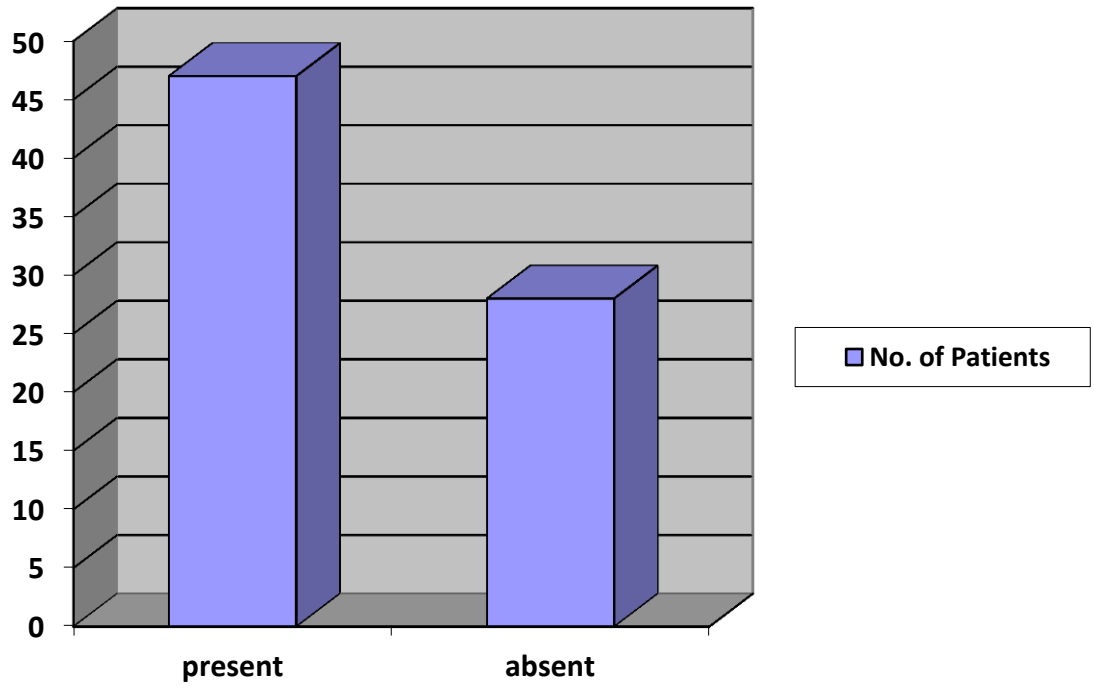
Frequency of Presence of free gas under Diaphragm

In patients with suspected perforative peritonitis mainly two types of X-rays were done i.e. X-Ray Erect abdomen and Chest X-ray PA view. In majority of the cases free gas under diaphragm was seen i.e. in 47 cases. out of the sample of 75 cases accounting for 62.67% of the cases.

TABLE 7 (PRESENCE OF FREE GAS UNDER DIAPHRAGM IN PATIENTS WITH PERFORATION)

FREE GAS UNDER DIAPHGRAM	FREQUENCY	PERCENTAGE
YES	47	62.67
NO	28	37.33

Chart 8 : Patients with free gas under diaphragm



Frequency of site of perforation along with the sex distribution in the Patients

(TABLE 8)

SITE	**SEX**		**TOTAL**
	MALE	**FEMALE**	
DUODENAL	44(65.67%)	3 (37.5%)	47(62.67%)
APPENDICULAR	9 (13.43%)	3 (37.5%)	12(16%)
GASTRIC	7 (10.45%)	NIL	7(9.33%)
ILEAL	3 (4.48%)	1 (12.5%)	4(5.33%)
JEJUNAL	1 (1.49)	1 (12.5 %)	2(2.67%)
ASCENDING COLON	1 (1.49)	NIL	1(1.33%)
RECTAL	1 (1.49)	NIL	1(1.33%)
SIGMOID	1 (1.49)	NIL	1(1.33%)
TOTAL	67	8	75(100%)

All the duodenal perforations observed in this study were anterior and none was posterior.

Frequency of computed tomography findings

CT was done in 28 of 75 patients in whom X-ray was inconclusive and the following finding are as follows

TABLE :9

SIGN	**FREQUENCY**	**PERCENTAGE**
Free fluid	15	53.57
Free air	9	32.14
Fat stranding	7	25
Air pockets	2	7.14

Thus positive findings were present in computed tomography in all cases with clinical suspicion of perforation with inconclusive results in plain radiograph.

Frequency of Postoperative Complications

Wound infection was found as the most important complications in the patients presenting with perforation accounting to be in 26 patients out of 75 , followed by URTI which was present in 10 patients.

COMPLICATIONS	FREQUENCY	PERCENTAGE
No complications	28	37.33
Wound infection	26	34.67
URI	10	13.33
LRI	15	20
Sepsis	9	12

TABLE 9 - Frequency of Postoperative Complications

Frequency of Operations Performed

Depending on the choice of the operating surgeon the procedure carried mainly omental patch repair was done for all Duodenal perforations and Gastric perforation , wheras simple perforation repair was done for Intestinal and Rectal perforations. Resection and anastomosis was done in a case of jejunal diverticulosis and ileal chronic ulcer suspicious of malignancy. Sigmoidectomy and Right hemicolectomy was done in case of sigmoid and ascending colon perforation respectively.

TABLE 10 - FREQUENCY OF OPERATIVE PROCEDURE DONE

OPERATIVE PROCEDURE	FREQUENCY	PERCENTAGE
Omental patch repair	54	72
Appendicectomy	12	16
Simple closure	5	6.67
Resection & Anastomosis	2	2.67
Sigmoidectomy	1	1.33
Right Hemicolectomy	1	1.33

TABLE 11 - DISTRIBUTION OF SAMPLE BY OUTCOME

OUTCOME	FREQUENCY	PERCENTAGE
Discharged	66	88
Expired	9	12

Out of 48 patients admitted with one day history of pain 3 died. Of the 23 patients admitted with 2days history of pain 3 died. All the patients admitted with 3 days history of pain died with 100% Mortality due to sepsis.

Out of 4 patients with Ileal perforation 2 patients died resulting in mortality rate of 50% in this study. Out of 47 patients with duodenal perforation 6 patients died resulting in 12.7% mortality rate in this study. out of 7 patients with gastric perforation 1 died with rate of 14.2% in this study.

Fig 5 . DUODENAL PERFORATION

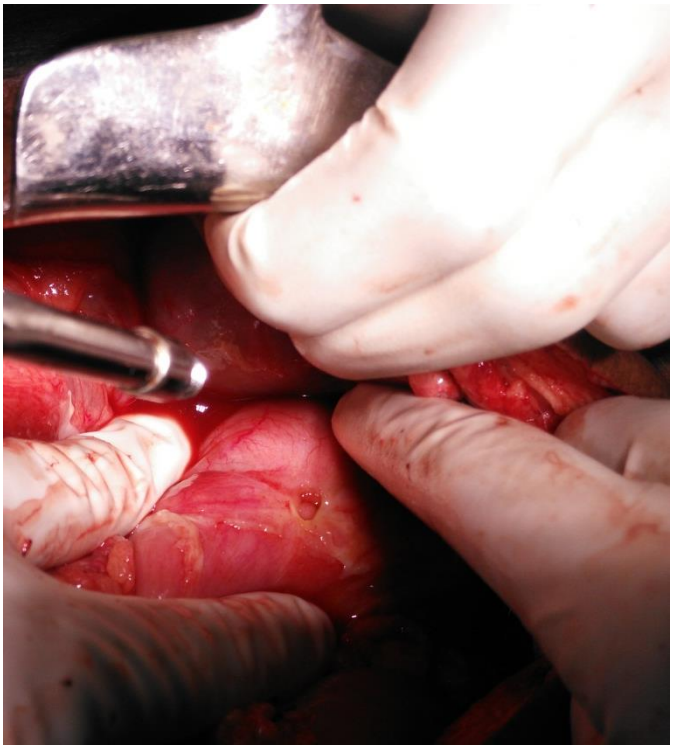


Fig 6. GASTRIC PERFORATION

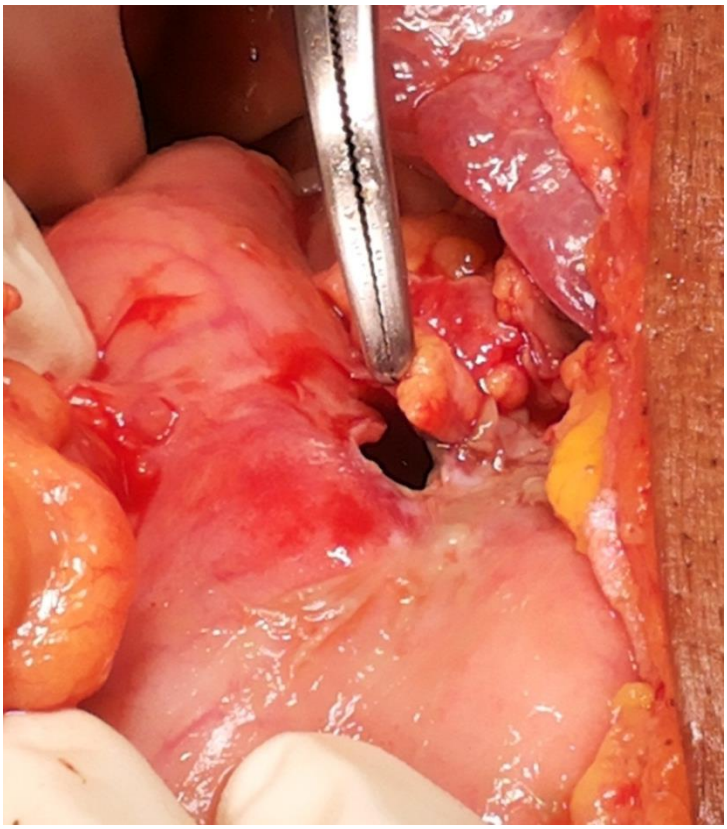


Fig 7. ILEAL PERFORATION IN TUBERCULOSIS

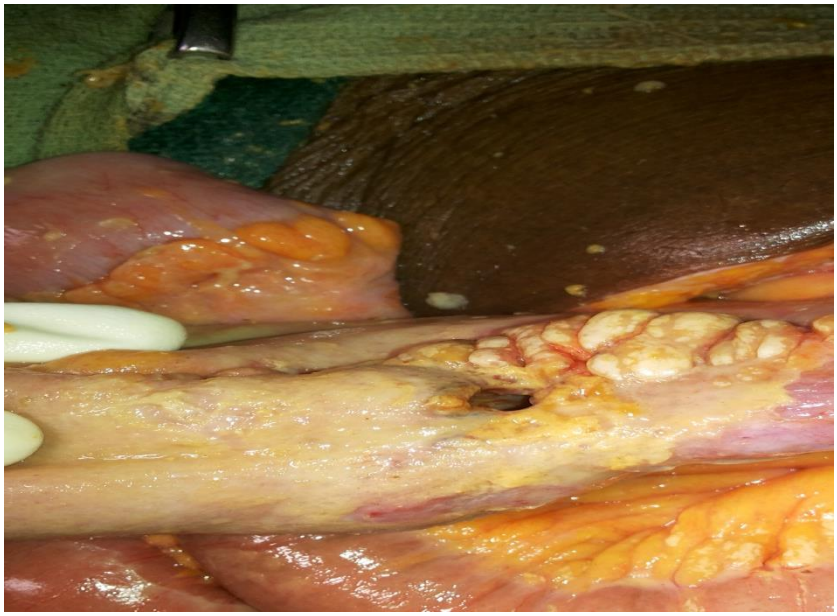


Fig 8 . ILEAL PERFORATION IN TYPHOID

DISCUSSION

This study was conducted in Thanjavur medical college hospital. A total of 75 patients admitted with particular criteria fixed during the study period were selected randomly.

The age distribution is as shown in Table 1. The highest number of patients encountered in this series were in the age group 50 years and above followed by the age group of 40 -49 years. In this present study, duodenal ulcer perforation was more common in the age group of above 50 years.

The ratio of men to women with all types of perforation irrespective of site and pathological condition was 9:1 in the present study. In the present study the number of male patients with duodenal ulcer perforation were 44 and the number of female patients with duodenal ulcer were 3. The number of male patients with appendicular perforation were 9 and female patients were 3. Ileal perforation was present in 3 male and 1 female cases.

The frequency of anatomical site involved in hollow visceral perforation is as shown in the table 3. The commonest site involved in this study was duodenal ulcer perforation (62.67%) followed by appendicular perforation (9%) and gastric perforation (7%)

In case of peptic ulcer perforations, pain abdomen and vomiting , Fever were the predominant symptoms. Tenderness, guarding rigidity, obliteration of the liver dullness were the predominant signs.

In the present study, pain abdomen was present in all cases. Vomiting was present in 56 0ut of 75 patients . Fever was present in 44 out of 75 patients. Guarding and rigidity was present in 47 out of 75 patients. Bowel sounds were absent in 84 % of patients at the time of presentation .

Absence of liver dullness was present in all cases of Gastric ,ileal and jejunal perforation. In 47 patients of duodenal ulcer perforation , liver dullness was obliterated in 36 patients of duodenal ulcer perforation. Liver dullness was not obliterated in 11 patients of duodenal ulcer perforation. This might be because of the sealing of the perforation or lack of gas at the site of perforation or adhesions around the site of perforations. Liver dullness obliteration was absent in all cases of appendicular perforation.

Diagnosis is made clinically and confirmed by the presence of pneumoperitoneum in radiograph. Free gas under diaphragm was present in 32 cases of duodenal ulcer perforation (68%), all cases of ileal perforation, jejunal perforation. one case of ascending colon and rectal perforation showed minimal free air in radiograph .

Computed tomography was done in 28 of 75 patients with clinical suspicion of peritonitis , with no free air in X Ray chest & abdomen erect. Free air was present in 9 cases of duodenal perforation which showed no free gas in X rays . Sealed air pockets were present in 2

cases of duodenal perforation. Free fluid helped in diagnosis in 15 out of 28 patients. Adjacent fat stranding noted in 7 out of 28 cases which were diagnosed as appendicular , sigmoid perforation intraoperatively.

Omental patch repair was done in all cases of duodenal and gastric perforation. Simple closure was done in 4 cases of ileal perforation and 1 case of rectal perforation. Resection and anastomoses was done in 2 cases of jejunal perforation due to diverticulosis. Ascending colon and sigmoid perforation were treated with resection, on postoperative follow up malignancy turned out to be the cause .

Wound infection 26 % was the most common post operative complication followed by lower respiratory tract infection 15% and upper respiratory infection in 10 % .

Death as a result of sepsis occurred in 6 duodenal ulcer patients , 2 cases of ileal perforation , 1 case of gastric perforation despite broad spectrum antibiotic coverage , management of fluid and electrolyte imbalance and intensive care. In this series overall mortality rate is 12 %.

CONCLUSION

- The most common age group affected is 50 years and above.
- Duodenal ulcer perforations were more common in the age group of 50 years and above.
- Most of these patients present with clinical signs of peritonitis 24 hours after the onset of pain.
- 89.33% of the patients were male and 10.67% of the patients were female.
- Duodenum (62.67%) is the most common site of perforation followed by appendicular perforation (16%) , gastric perforation (9.33%) and enteric perforation (8%).
- Guarding and rigidity was present in 62. 67% of patients.
- Diagnosis is made clinically and confirmed by presence of free air under diaphragm in 62.67 % of patients
- Computed tomography aided in diagnosis in all (100%) cases with no free air in x rays.
- Laparotomy with closure of the perforation with omental patch (72%) is the commonest operative management for perforated peptic ulcer.
- The most common postoperative complication observed was wound infection and lower respiratory tract infection.
- The mortality rate observed in this study were 12.7%, 14.2%, 50%

Duodenal , gastric and ileal perforation respectively. The overall mortality rate is 12%.

SUMMARY

Duodenum was the most common site of perforation in perforative peritonitis due to hollow viscus perforation. The highest number of patients was seen in the age group 50 years and above, irrespective of the pathological conditions followed by 40-49 year age group. Most of the patients presented within 24 hours after onset of the clinical symptoms. Duodenal ulcer perforation was the most common cause of perforation in perforative peritonitis due to hollow viscus perforation, next commonest was appendicular followed by gastric and enteric perforation .

X rays alone is diagnostic in 62. 67% of patients with perforative peritonitis. Computed tomography aided in diagnosis in the remaining patients . Laparotomy with closure of the perforation with omental patch closure is the commonest method of surgical management in perforative peritonitis due to hollow viscus perforation. Wound infection and Lower respiratory tract infection is the most common complication observed. Mortality was more in cases of peptic ulcer perforation with more than 48 hrs of pain , enteric perforation and very low in appendicular perforation .

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PROFORMA

Case No. IP No. Unit

Name: Age: Sex

Date of admission:

Date of operation:

Date of Discharge / Death:

1. COMPLAINTS:

a. Pain Abdomen

i) Duration ii) Mode of onset

iii) Site of pain IV) Nature

v) Radiation VI) Shifting of pain

vii) Aggravating / Relieving factors

b. Vomiting

i) Duration ii) Frequency

iii) Vomitus: Bilious / Blood / Faecal / otherwise.

c. Fever

i) Duration ii) Degree

iii) Chills / Rigors

d . Distension of abdomen

i) Present / absent:

ii) Duration (if present):

e. Bowel symptoms.

i) Diarrhoea ii) Constipation

iii) Hematemesis/ Melaena

f. H/o reduced / nil urine output

g . other complaints (if any)

2. PAST HISTORY:

a) H/o previous surgery

b) H/o Trauma

c) Any comorbid illness : DM / HT / TB /COPD

3 . PERSONAL

a) Diet : Mixed/vegatarian

b) Habits : Smoker

Alcoholic

GENERAL PHYSICAL EXAMINATION:

Vital signs:

Pulse: Blood Pressure:

Temperature: Respiratory rate

State of hydration

Anaemia / Jaundice / Pedal edema / Lymphadenopathy

6. EXAMINATION OF THE ABDOMEN

a) Inspection

1. Shape 2.Umbilicus

3. Movement with respiration

4. Hernial orifices 5. External genitalia

6. Distension

b) Palpation:

1. Site of tenderness 2. Guarding / Rigidity

c) Percussion :

Liver dullness obliteration

Free fluid

d) Auscultation

Bowel sounds: Y/N

7. EXAMINATION OF OTHER SYSTEMS

a. Cardiovascular system

b. Respiratory system

c. Nervous system

8. INVESTIGATION

Blood investigations:

1.Hb% TC Blood Group

BT

CT

2. Urine Sug Alb Micro

3. RBS, Urea , Creatinine

4.Special: X-ray abdomen (Erect / lateral decubitus)

X-ray Chest PA view

5. USG abdomen

6. CT abdomen & pelvis

9. CLINICAL DIAGNOSIS

10. FINAL DIAGNOSIS

11. COMPLICATIONS

12. OUTCOME.

KEY TO MASTER CHART

A	-	Absent
AP	-	Appendicular perforation
ACP	-	Ascending colon perforation
AR	-	Air Pocket
BS	-	Bowel sounds
D	-	Diffuse
DIS	-	Discharge
DOP	-	Duration of pain
DP	-	Duodenal perforation
E	-	Epigastric
EL & A	-	Emergency laparotomy and appendicectomy
EXP	-	Expired
F	-	Fever
FF	-	Free fluid
FGD	-	Free gas under diaphragm
FA	-	Free Air
FS	-	Fat stranding
G&R	-	Guarding and Rigidity
GP	-	Gastric Perforation

HG - Hypogastrium

IP - Ileal perforation

JP - Jejunal perforation

LRI - Lower respiratory tract infection

LDO - Obliteration of liver dullness

N - No

OPR - Omental patch repair

P - Present

R&A - Resection and Anastomosis

RH - Right Hemicolectomy

RIF - Right iliac fossa

RP - Rectal Perforation

SC - Simple closure

SEP - Sepsis

SOT - Site of tenderness

SP - Sigmoid perforation

URI - Upper respiratory tract infection

V - Vomiting

WI - Wound infection

Y - Yes

CONSENT FORM

Name : Date :
Age : Sex :

I have been explained clearly about the research and its objectives. I understand the facts and I give full consent to be included as a participant in the " A CLINICAL STUDY OF PERFORATIVE PERITONITIS."

 I have been explained about the nature of the study,clinical and laboratory investigations required for this study.

 I have been explained about my rights and responsibilities by the investigator.

 I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the regulatory authorities, Govt. agencies, and IEC.

 I have understood that my identity will be kept confidential .

 I have my questions being answered to my satisfaction.

 I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Date : Signature of the patient

SL.NO	NAME	AGE	SEX	IP.NO	DOP	SOP	VO	F	G&R	LDO	BS	XRAY FGD	CT ABD	DIAGNOSIS	PROCEDUR	COMPLICA	OUTCOME
1	MARIYAPP	70	M	19258	3	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	SEP	EXP
2	PANEERSEL	70	M	19469	1	RIF	Y	Y	Y	Y	A	Y	NA	ACP	RH	LRI,WI	DIS
3	KALIYAPER	55	M	20150	1	D	N	Y	Y	Y	A	Y	NA	GP	OPR	URI	DIS
4	ABHINANTI	26	M	19345	1	RIF	Y	N	N	N	A	N	FS	AP	ELA	N	DIS
5	KANNAN	30	M	20253	1	D	N	N	N	Y	P	Y	NA	DP	OPR	WI	DIS
6	CHIDAMBA	40	M	21336	1	E	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
7	MATHI	40	M	19709	1	E	Y	N	Y	Y	A	Y	NA	GP	OPR	N	DIS
8	BALA	43	M	20573	2	D	N	N	Y	N	A	N	FA,FF	DP	OPR	URI	DIS
9	TAMILARAS	40	F	20932	1	D	Y	N	N	N	P	N	FA	DP	OPR	WI	DIS
10	RENGASAM	52	M	21444	3	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	SEP,LRI	EXP
11	SHANMUG	57	M	20679	1	D	N	Y	N	Y	A	Y	NA	DP	OPR	WI	DIS
12	RAJAPPAN	50	M	20738	1	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
13	GAUTAM	45	M	20962	1	D	Y	N	Y	N	A	N	FA,FF	DP	OPR	WI	DIS
14	RAJASEKAR	32	M	20809	1	E	Y	N	Y	Y	A	Y	NA	GP	OPR	N	DIS
15	NARESH	19	M	22174	1	RIF	Y	Y	N	N	P	N	FF	AP	ELA	WI	DIS
16	SANGILIML	62	M	21762	1	E	Y	Y	Y	Y	A	Y	NA	GP	OPR	SEP,LRI	EXP
17	THANGAIY	50	M	22648	1	D	Y	Y	Y	Y	A	N	AR	DP	OPR	LRI,WI	DIS
18	CHIDAMBA	75	M	24161	1	E	Y	N	N	Y	A	Y	NA	DP	OPR	WI	DIS
19	RATHINASA	60	M	24291	1	D	Y	N	N	Y	P	Y	NA	IP	RA	LRI	DIS
20	VADIVEL	60	M	11356	2	D	Y	Y	Y	N	A	N	FA	DP	OPR	URI	DIS
21	ARULPAND	24	M	14073	1	D	Y	Y	Y	Y	P	Y	NA	DP	OPR	N	DIS
22	PALANIYAP	43	M	18996	2	HG	Y	N	N	N	P	Y	NA	RP	SC	LRI,WI	DIS
23	KATHAIYAM	53	M	25898	1	D	Y	N	N	N	P	N	FA	DP	OPR	N	DIS
24	VENILLA	44	F	23853	1	RIF	Y	Y	N	N	A	N	FF	AP	ELA	WI	DIS
25	KUPPUSAM	30	M	13616	1	E	Y	N	N	Y	P	Y	NA	DP	OPR	N	DIS
26	KALAIYARA	20	M	15481	1	D	Y	N	Y	N	A	Y	NA	DP	OPR	N	DIS
27	CHELLADUI	54	M	17520	3	D	Y	Y	Y	Y	A	Y	NA	IP	RA	LRI,SEP	EXP
28	BOOPATHI	17	M	18160	2	RIF	Y	Y	N	N	A	N	FF	AP	ELA	WI	DIS
29	DAHANABA	63	F	15538	2	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	LRI,SEP	EXP
30	CHANDRAM	57	M	14837	1	D	Y	N	N	N	P	N	FA,FF	DP	OPR	URI,WI	DIS
31	PERIYASAM	43	M	13558	1	D	Y	Y	Y	N	A	Y	NA	DP	OPR	N	DIS
32	RAMAIYA	56	M	13735	2	D	Y	Y	Y	N	A	N	FA	DP	OPR	LRI,SEP	EXP
33	RAJENDRAI	54	M	12911	1	RIF	Y	Y	N	N	P	N	FS	AP	ELA	WI	DIS
34	SUBASH	28	M	12627	1	E	N	N	Y	N	A	N	FA,FF	DP	OPR	LRI,SEP	EXP
35	SUGUNA	19	F	18657	2	RIF	Y	N	N	N	P	N	FF	AP	ELA	N	DIS
36	KUMAR	42	M	15896	1	D	N	N	Y	Y	A	Y	NA	DP	OPR	N	DIS
37	VEERAIYAN	35	M	13362	2	RIF	N	N	N	N	A	N	FS	AP	ELA	WI	DIS
38	KUNJU	70	M	12369	1	E	Y	N	Y	Y	A	Y	NA	DP	OPR	URI	DIS
39	SUBRAMAN	48	M	16420	2	RIF	N	Y	N	N	A	N	FF	AP	ELA	WI	DIS
40	GUNASEKA	55	M	13439	2	D	Y	Y	Y	N	A	Y	NA	DP	OPR	N	DIS
41	KUPPUSAM	30	M	13616	1	E	Y	N	Y	N	A	Y	NA	DP	OPR	N	DIS
42	RAJAGOPA	34	M	12911	2	D	N	Y	Y	Y	A	Y	NA	DP	OPR	URI	DIS
43	KAVIARASU	35	M	13362	2	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
44	SUBRAMAN	33	M	16264	1	E	Y	N	N	N	A	N	FF	DP	OPR	URI	DIS
45	TAMILARAS	40	F	28489	1	D	Y	Y	Y	N	A	Y	NA	DP	OPR	N	DIS
46	AYYAPAN	27	M	28692	1	RIF	Y	Y	N	N	A	N	FS	AP	ELA	WI	DIS
47	JEEVA	28	M	31038	1	RIF	Y	Y	N	N	A	N	FF	AP	ELA	WI	DIS
48	JEYARAMA	52	M	35355	1	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
49	SATISHKUM	14	M	36931	1	E	Y	N	Y	N	A	Y	NA	DP	OPR	N	DIS
50	BALAIYAN	35	M	27442	2	E	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
51	VALLUVAR	45	M	30062	1	RIF	N	Y	N	N	A	N	FF	DP	OPR	WI	DIS
52	MARUDAM	65	M	30623	2	E	Y	N	Y	Y	A	Y	NA	DP	OPR	N	DIS
53	SAMIAYYA	65	M	34420	2	D	N	Y	Y	Y	A	Y	NA	DP	OPR	URI	DIS
54	RASU	73	M	31601	2	D	N	Y	Y	N	A	Y	NA	DP	OPR	URI,SEP	EXP
55	ARIVALAGA	38	M	33468	1	E	N	N	Y	N	A	Y	NA	DP	OPR	WI	DIS
56	RAM	48	M	34142	1	E	Y	Y	Y	Y	A	Y	NA	GP	OPR	WI	DIS
57	SHAHJAHA	55	M	30964	1	RIF	N	N	N	N	P	N	FF	AP	OPR	WI	DIS

58	GOVINDAS	70	M	36802	1	DN	N	N	N	N	A	N	FA	DP	OPR	LRI	DIS
59	SUDARSAN	65	M	39602	1	RIF	Y	Y	N	N	A	N	FS	AP	ELA	WI	DIS
60	ARUNLAL	25	M	33335	1	E	Y	N	Y	Y	A	Y	NA	IP	RA	N	DIS
61	KATHAYEE	65	F	33771	3	D	N	Y	Y	Y	A	Y	NA	IP	RA	SEP,LRI	EXP
62	KALYANASI	55	M	35927	1	D	N	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
63	KALYANAN	55	M	35967	2	D	Y	Y	N	N	A	N	AR	DP	OPR	WI	DIS
64	GOVINDAS	70	M	36802	2	D	Y	Y	Y	Y	A	Y	NA	IP	RA	LRI,WI	DIS
65	PITCHIYAN	19	M	29797	1	E	Y	Y	Y	Y	A	Y	NA	DP	OPR	N	DIS
66	NEELAKAN	17	M	29295	1	D	Y	N	Y	Y	A	Y	NA	DP	OPR	N	DIS
67	PARAMESH	55	F	28737	1	D	N	Y	N	N	A	Y	NA	JP	RA	WI	DIS
68	MARIMUTH	60	M	30589	1	D	Y	Y	N	N	A	N	FF	DP	OPR	WI	DIS
69	KRISHNAN	66	M	38224	2	D	Y	Y	Y	N	A	Y	NA	JP	RA	N	DIS
70	NAGAMMA	40	F	43376	1	RIF	Y	N	N	N	A	N	FS	AP	ELA	WI	DIS
71	KATHIRVEL	66	M	43476	2	E	N	Y	Y	Y	A	Y	NA	GP	OPR	N	DIS
72	MANIKAND	32	M	21412	2	D	Y	Y	Y	Y	A	Y	NA	DP	OPR	LRI	DIS
73	PALANIYAP	44	M	40301	1	E	Y	N	Y	Y	A	Y	NA	DP	OPR	N	DIS
74	DURAIRAJ	76	M	43806	2	LIF	Y	Y	Y	N	A	N	FS,FF	SP	SIG	LRI,WI	DIS
75	RAJASEKAR	32	M	38233	1	E	Y	N	Y	Y	A	Y	NA	G	OPR	LRI	DIS